GLAUCOMA;

ITS SYMPTOMS, VARIETIES, PATHOLOGY AND TREATMENT.

STIRLING.

GLAUCOMA;

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PREFACE.

The contents of this volume were put together in great part in connection with my lectures to the students of the Post-graduate Medical School and Hospital, New York. They were published serially in the ANNALS OF OPHTHALMOLOGY, and on the recommendation of friends in the profession are now, with considerable diffidence, reproduced in book form, in the hope that they may, perhaps, prove a saving of some labor to those interested in the history of the subject of glaucoma.

I desire especially to express my indebtedness to Mr. Treacher Collins, through whose friendly interest I was enabled to carry out my observations in the Moorfield's Laboratory on the condition of the vortex veins and choroid in this disease, and for much other kindness in connection with pathological work there. I am also indebted to Dr. Katherine Collins for photographing such of my sections of glaucomatous eyes as are here reproduced.

ALEX. W. STIRLING.

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ERRATA AND ADDENDA.

Page 49 line 17 from bottom *of* should be *to*.

" 56 " 2 " " 75 " ".75.

" 66 the figure should be on page 94.

" 78 add to line 6 from top: Martin of Bordeaux asserts that astigmatism "contrary to the rule." *i. e.*, with the meridian of greatest curvature horizontal, is found in 50 per cent. of all cases of glaucoma, which has been corroborated by Pfalz; and in certain cases of glaucoma increase or diminution of the grade of astigmatism goes hand in hand with increase or diminution of tension. (Javal in the Norris and Oliver System of Diseases of the Eye, Vol. 2, page 128, 1897.) This statement may be compared with that on page 118 of this book.

Page 81 line 18 from bottom *axis* should be *axes*.

" 107 " 8 " top *without* should be *with*.

" 109 " 8 " bottom *result* should be *results*.

" 161 " 1 " top *Knies'* should be *Knies's*.

" 161 " 2 " top *Vincentiis's* should be *Vincentiis'*.

Page 168 add to line 12 from bottom: Dr. Nicati has been kind enough to send me recently a note relative to the conclusions at which he has arrived concerning the worth of the various operations for glaucoma. He places first iridectomy ("sclero-iridectomy"), which facilitates by the sclerotomy the flow of the aqueous secretion, and by the iridectomy weakens "spasms of the choroid" (see Nicati's theory), while at the same time the retention of watery secretion and sudden elevations of tension by choroidal œdema are limited. Even outside of cases of pupillary occlusion, it is the true physiological operation for glaucoma. After iridectomy the next most efficacious operation is scleriritomy which adds to the sclerotomy a section of the iris, true neurotomy, which acts like an excision of the iris, but less permanently. Recurrences he now knows to be more frequent than after iridectomy, but it is the preferable proceeding for the prevention of the formation of staphyloma after total adherent cicatricial leucoma. He frequently operates from without inwards by

means of a long cataract knife introduced under the conjunctiva, and used with a sawing motion until the aqueous secretion flows. In the third place he puts the sclerotomy of Quaglino and Wecker, and in the fourth puncture of the vitreous, or the equatorial sclerotomy of MacKenzie, whose effects are quite transitory owing to fibrous transformation of the vitreous humor in the fistula.

GLAUCOMA; ITS SYMPTOMS, VARIETIES, PATHOLOGY AND TREATMENT.

CHAPTER 1.

SYMPTOMS AND THEIR EXPLANATION.

(The figures in the text refer to the bibliographical list at the end of each chapter.)

Glaucoma, which forms about one per cent of all the diseases of the eye (Fuchs, 1), is a subject of great interest to the ophthalmologist, an interest which ought to be shared by all practitioners of medicine, because, unfortunately, its symptoms so closely simulate other diseases, that it is by no means an uncommon thing that their true origin is overlooked, with the unhappy result that valuable time is wasted, or improper remedies applied; so much so, indeed, that vision has frequently gone beyond recall before a correct diagnosis has been made. This is a condition of matters which every ophthalmic surgeon of any experience has had frequently to deplore, for a tentative diagnosis is usually easy without an ophthalmoscope, and all that is wanted is that the practitioner bear in mind, firstly, that there *is* such a disease as glaucoma, whose symptoms may much resemble rheumatism and neuralgia of the head and face, including the teeth and ear, sick headache, as well as some common forms of inflammation of the eyes, and that it is sometimes attended by fever and vomiting; secondly, that atropine *is not* a panacea for every ocular ill, but on the contrary is harmful in many, especially in glaucoma, and should never be used except with full understanding of its action in the disease for which it is employed; and thirdly, that he make himself acquainted with the resistance, or "tension," of healthy eyes when palpated as for an abscess, and remember that in glaucoma this tension is greater than normal.

If attention be paid to these points glaucoma will seldom go undetected, for, in chronic cases, in which all active symptoms and high tension may be in abeyance, the visual symptoms draw the attention of the patient and the surgeon in the right direction.

In an acute case the painful symptoms frequently begin at night, and the first visual discomfort is frequently the appearance of halos, rainbows, or rings of colored light, surrounding the flame of gas or lamp, while objects seem to be enveloped in a fog, and the field of vision is diminished.

The lids are now discovered to be red and swollen, the conjunctiva injected, even chemotic, the anterior ciliary veins enlarged and forming a dull red circle around the cornea, which itself is hazy, possibly superficially uneven, especially at the center, causing all behind it to be dimmed or altogether invisible, while it is more or less insensitive to touch. The pupil can, however, usually be seen to be enlarged, perhaps irregular, and then most often is oval with the long axis vertical, and presenting that greenish hue from which the disease received its name. The iris reacts slightly and slowly, or even not at all. It seems to have lost its brilliance and something of its color, while its finer markings may have disappeared.

The anterior chamber, between the iris and cornea is shallower than normal, and when the eye is palpated between the fore-fingers it is found to be too hard.

On the basis of these observations a diagnosis of glaucoma can safely be made, but the surgeon will naturally desire to see what the ophthalmoscopic picture is. If the condition of the cornea permit, he will find the other media clear, or the aqueous perhaps a little cloudy. On the disc, the veins will be enlarged and tortuous, probably pulsating, the arteries small, and possibly pulsating; and besides at this stage probably nothing remarkable will be observed. Such is the eye during an acute attack, but it would be a mistake to look upon this as the only type of the glaucomatous condition. On the contrary, such attacks are usually intermittent, besides which there is often an initial, or "prodromal" stage, lasting any length of time from days to years, during which the eye is subject at intervals, at first often of months, later maybe of days, to symptoms resembling the above attacks, but milder, and limited often to halos and slightly foggy vision, with perhaps a little pain and congestion. Each of these abortive attacks, which are especially apt to appear during fatigue, hunger, or anxiety, soon passes away, perhaps after the first sleep or meal, vision returning to its normal, except that difficulty of accommodation may become abnormally apparent, presbyopic glasses being frequently exchanged for stronger ones. When the prodromal stage has passed by gradual accentuation into the acute, and this has become "confirmed" by the continuation of the attacks, the eye is found to be deteriorating, never quite free from congestion with raised tension, and a vision which does not return to the normal between attacks, and after each one becomes less and less acute.

It is now that a condition of the disc, important from the points of view of both pathology and diagnosis, and which has not yet been touched upon, is in all likelihood to be discovered—the glaucomatous cup or excavation, a depression of the entire optic nerve head which has usually lost something of its color and become atrophic looking.

The third stage, called "glaucoma absolutum," is reached when, having continued to deteriorate till the field has first been reduced to merely indifferent central vision, and then that also having been destroyed, the eye has finally become quite blind, though probably without cessation of discomfort, for it may for long continue inflamed and hard, but holding out false hopes to the patient's mind, through the flashes of light, "photopsia," mere subjective symptoms, that vision may yet be recovered. The whiteness of the sclera now tends to take on a bluish tint, edged around the cornea by the red circle of the distended ciliary veins; the cornea remains insensitive, possibly more or less opaque, and even roughened externally by epithelial ulcers, which may also extend more deeply into its substance. The anterior chamber is shallow; the pupil still large; irregular, and greenish; and the small circle of greyish iris is bordered at the pupil by a ring of black pigment. The globe is very hard, and the disc is deeply excavated. At this stage Brailey (2), Pagenstecher, etc., have observed the excavation fill up again with new tissue.

The further changes through which the eye passes are those of degeneration. As the softening tissues give before the internal pressure, the globe tends to become square, bulging between the recti muscles, and staphyloma may also form at the equator: in the region of the ciliary body, with the anterior ciliary vessels visible in front of it, "Ciliary Staphyloma"; or between the ciliary body and the cornea, "Intercalary Staphyloma," when these vessels lie behind it. The cornea also becomes opaque, and perforation may result from ulceration, and lead to iridocyclitis, panophthalmitis, and phthisis bulbi. The lens usually becomes cataractous, and sometimes calcareous. Secretion ceasing with the destruction of the ciliary region, tension then goes down, until the globe becomes quite soft and shrinks, when first the patient obtains relief.

Instead of passing through the comparatively long stages as described above, the disease may take not more than a few hours or even less to carry an eye from normal vision to total blindness. "Glaucoma Fulminans," which affords a striking contrast to another form, "Glaucoma Simplex," in which the destruction of the eye drags slowly through a period, sometimes of many years, with little or no painful or inflammatory symptoms, and marked, maybe, alone by gradual deterioration in acuity of central vision, and in the dimensions of the field. But

not uncommonly these chronic cases have seasons of accentuation when halos are seen and vision is more distinctly dimmed. Though the cornea is generally clear the ciliary veins are frequently enlarged; the anterior chamber may or may not be shallow; the iris may or may not be of normal color, size, and shape, and may react well or indifferently. In short, the anterior segment of the globe may appear normal, or may present more or less of the symptoms to be found in acute cases.

When the tension and anterior parts of the eye give only an indefinite clue or none, to the true nature of the disease, the diagnosis rests chiefly upon the condition of the disc and upon certain visual symptoms.

Between the two extremes, glaucoma fulminans and the quietest form of glaucoma simplex, the interval is completely filled by other types constituting a continuous chain, at no point in which can lines of absolute demarcation be drawn, to form definite divisions according to acuity. In the majority of cases, both eyes are affected, either simultaneously, or the second follows the first at an interval which may extend even to years.

Secondary Glaucoma.

Certain diseased conditions of the eye have a causal relationship to glaucoma which will be discussed more fully later on. Among these are haemorrhagic retinitis, intra-ocular tumors, iris bombé, anterior synechia, swollen or dislocated lens, congenital malformations, etc.

Explanation of Symptoms.

It is held by the majority of authors that the other symptoms of glaucoma are secondary to the increased tension, or that increased tension and glaucoma are practically synonymous; but, as will be seen later, some surgeons believe that the optic nerve changes begin before the appearance of *plus* tension, and some that glaucoma simplex may run its course with normal tension, while others consider that in these cases the tension is relatively high because the lamina cribrosa is unnaturally weak. How the tension is increased, in the first place, will be discussed under the heading of etiology; but, having once appeared, its effects soon follow, and, on its reduction to normal, they speedily disappear, those, at least, which are not due to changes which, through the continuance of the high tension, have become of a necessarily permanent character. The higher and the more sudden the abnormal tension the more intense are the symptoms. Its effects have been compared (Critchett, Priestley Smith, 3, Fuchs, 4) to those of an incarcerated hernia

The Swelling of the Lids and Conjunctiva

present in very acute cases is probably the result of blood stasis

in vessels in the neighborhood of the globe, due to the reaction of the vaso-motor nerves to the irritation of the sensory nerves of the eye, and resembling that produced by a foreign body in the orbit.

The Enlargemont of the Anterior Ciliary Veins

is due to the pressure on the vortex veins, which pass obliquely through the sclerotic closing them to some of the fluid which should leave the eye by their channels, but is forced instead to take the route by the anterior ciliary veins.

The Hazy Condition of the Cornea

was first described by Liebriech in 1863, and was supposed by many (e. g., by Mauthner, 5) to be due to keratitis; but this supposition may be set aside on account of the rapidity of its disappearance on reduction of the intra-ocular tension. Both Arlt & Leber had already ascribed it to oedema, when Fuchs (6) described the spaces between the corneal lamellae, and chiefly anteriorly, as infiltrated by a coagulated fluid, which penetrated also to Bowman's membrane, and surrounded the nerve filaments which reach to the superficial epithelium. If this corneal oedema were due, as suggested by Knies and Weiss after experiments with chemical agents to the passage of the aqueous humor under the influence of pressure into the corneal tissue, we should not, as Treacher Collins (7) suggests, expect to find the posterior layers less affected than the anterior, as is actually the case. It is more probably due to interference with the circulation of the nutrient fluid of the cornea which tends to collect towards its center.

The Halos or Rainbows

Seen in the early stages, during short intermittent attacks, have certain constant characteristics. The outer ring is always red and the inner is bluish; the former more visible around gas and candle light, the latter around the electric light. Between the inner ring and the light is usually a clear space, but occasionally having lines radiating through it. The farther away the light the larger the halo, which, by the perimeter, measures from seven degrees (Donders) to ten or eleven degrees (Laqueur). Rarely a second halo has been seen outside the first. Mauthner (8), though he saw the possibility of the corneal oedema causing the rainbows, was inclined to think them due to nerve irritation, as was also Dobrowolsky (9) after experimentally producing congestion of his own head and eyes. Donders (10) was the first to produce some proof, in 1850, that they are dependent upon an actual lesion of one or other of the ocular tissues. This he did by showing that on covering the lower half of the pupil, the halo disappears in the superior external and inferior internal quadrants, and the reverse when the upper

half is covered; also, that the halo remains in the same position when the eye looks some distance from the light. The dilatation of the pupil and alterations of the lens when accommodating, he thought had some influence in the production of this symptom.

De Wecker attributed it to slight changes in the corneal epithelium, due to temporary increase of pressure; and Treacher Collins (11), in a review of the subject, relates experiments by which, with a 0.125 per cent. solution of hydrochlorate of erythrophloeine he had produced, while the tension remained normal, a condition of the cornea exactly resembling in appearance that found in early glaucomatous tension, and associated with very slight corneal insensibility with blurred vision, and precisely similar halos. By its means he has shown that these halos have no connection with the size of the pupil, with the refraction of the eye, or with the condition, or even the presence, of the lens. Still, that the lens may give rise to halos is asserted by Homolle and Quevenne, and Lauder Brunton (12) produced them by the use of digitalin, a result he believed, of a slight opacity of the lens. Cocaine also produces an opacity of the cornea, which is, however, unassociated with halos, because, according to Treacher Collins, it is too intense, and corresponds with the later stage of glaucomatous oedema when the halos have passed away. The writer has frequently observed "glaucomatous halos" follow immediately upon the use of a lotion of boric acid and sulphate of zinc. Wurdinger (13) after experiments on rabbits with cocaine and fluorescine, says that the former causes shrinking of the whole cornea; that in the late stages, epithelial cells are cast off in portions of it; and Thomalla (14) further proves the resemblance between the glaucomatous and cocaine opacities, by showing that, in acute glaucoma, fluorescine always, in his experiments, colored some portion of the cornea.

Priestley Smith, before Collins made his experiments, thought that the halos were due to "the latent physiological aberration being in some way rendered manifest or exaggerated," but now (15) he is satisfied that they should be ascribed to the peculiar condition of the corneal epithelium described above. In mild iritis (Schweigger 16) from the pressure of a film of secretion upon the cornea in conjunctivitis, and in normal eyes sometimes when the pupil is dilated (Berry 17), halos have been found.

The Insensibility of the Cornea

was ascribed by Fuchs (18) in 1882, to œdema of nerve endings, and in 1893 (19) he says of it, "through the high intra-ocular pressure the ciliary nerves are

compressed and paralyzed; in this way the insensibility of the cornea comes about."

The Iris.

A change in the hue of the iris, and any obliteration of its delicate markings which may be found in early glaucoma, have been considered by many (as by Mauthner (20) in 1878, and Brailey 21, in 1881), to be due to actual inflammation; but that opinion is not now so commonly held, but rather that these are due to simple hyperaemia, the result of venous stasis, and producing rigidity and oedema of the iris, and occasionally even haemorrhages (Schnabel (22), Treacher Collins (23), Fuchs (24). At the same stage the comparative immobility of the iris, and the large size and irregularity of the pupil, are usually considered to be due to paresis of nervous fibres within the eye. Schweigger (25) remarks, "that the ciliary nerves are very sensitive to pressure is proved by the occurrence of mydriasis traumatica which may follow contusions of the eye which leave behind them no bad effect." By others the condition is ascribed to constriction of the vessels entering the iris. Objections might be urged to each of these theories, and probably the results are not always due to the same cause. Thus: there is no real paralysis, because the iris reacts to both eserine and atropine. Then why should the pupil dilate when the ciliary nerves are pressed upon? The sympathetic fibres must receive a compression equal to that upon those of the third nerve; or, does this merely go to show that when nerve influence is altogether removed from, (for paralysis rather than stimulation is indicated by the long continuance of the pupillary dilatation) or equally restricted in, the various nervous fibres of the iris, the position of rest is that of dilatation such as occurs in advanced poisoning by chloroform?

On the other hand, we know that hyperaemia of the iris, as in the early stages of iritis, produces contraction of the pupil, while anaemia of it, as caused by cocaine, produces dilatation. In glaucoma we have a combination of hyperaemia and dilatation.

The oval shape of the pupil is capable of explanation on the ground that the iris in glaucoma is frequently, by the ciliary processes, pressed against, or is even adherent to, the back of the cornea, and not usually equally so at all points of its circumference. At the later stages the iritic changes are of a different type, for then the vessels have been in a great part obliterated by long-continued pressure, with secondary atrophy of the true tissue, and to some extent replacement of it by new connective tissue fibres. The black border at the pupillary margin, "Uveal Ectropion," is due, according to Knies (27), to the organization of inflammatory material on the front of the iris, pulling the uveal pigment round to the anterior surface;

but Treacher Collins (28) ascribes it to shrinking of the stroma of the iris more markedly than of the pigment on its posterior surface.

The Color of the Pupil

which gave origin to the name of glaucoma (from the Greek for sea-green) as well as to its old title of "green cataract," is produced by the reflection of the light entering the lens, modified by the state of the cornea and aqueous humor. It is not peculiar to glaucoma, and is seen in other conditions where dilated pupil and imperfectly transparent media are associated.

The shallowness of the anterior chamber is in primary cases, no doubt, usually due to the excessive pressure behind the lens, zonule, and iris, which brings the iris into closer apposition with the cornea, but as Priestley Smith (29) remarks, it is not necessarily so, for the lens may be of excessive thickness from a natural growth or from cataractous swelling. In some secondary cases, it is caused by a dragging forward of iris, ciliary processes, lens, or vitreous, by adhesions between these and the cornea. There are certain cases to be discussed later in which the anterior chamber is not shallow.

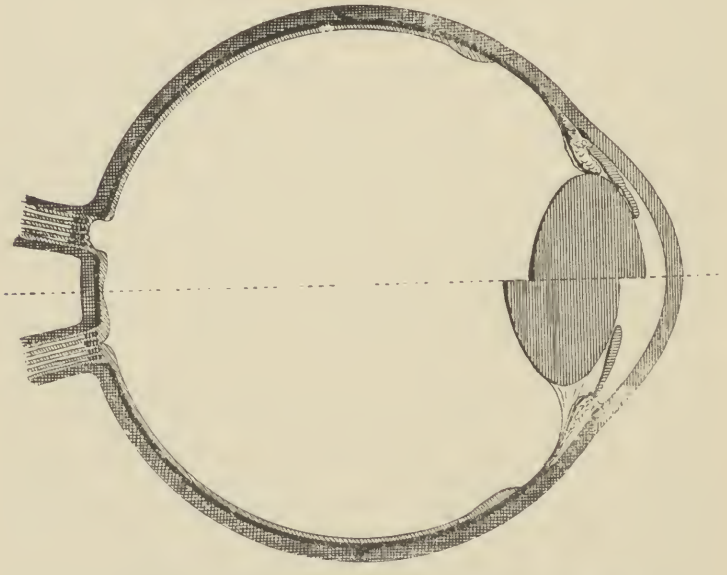

FIG. 1.—Diagram comparing the Normal with the Glaucomatous Eye.

The Menia.

At the beginning of a glaucomatous attack, unless when associated with uveal inflammation, the aqueous, lens, and vitreous are all clear, but soon on account of exudation from enlarged vessels, the aqueous and vitreous may lose their per-

fect transparency. In the late stages the lens, like other parts of the eye, degenerates, and this form is to be distinguished from an ordinary simple or traumatic cataract appearing in a glaucomatous eye. This may be done, according to Fuchs, because the former shows a more noticeable swelling, a bluish white color, along with a bright silky sheen.

The Enlargement and Tortuosity of the Retinal Veins

is explained by the difficulty experienced by the blood in returning through vessels which are specially exposed to pressure where they bend into the optic nerve and pass through the lamina cribrosa.

The Pulsation of the Retinal Veins

was described by Van Tricht of Utrecht in 1853 and in the same year by Coccius; and Donders concluded, from his investigation of the subject, that this pulsation is normal in all eyes and at all ages. As a matter of fact by careful observation of the vein upon the disc, at which point its internal pressure is lowest, and especially after it has bent into a physiological cup, the pulsation can be very frequently seen, and Lang and Barrett (30) found it in 73.8 per cent. of the eyes examined by them at Moorfields. It may be present at one time and not at another in the same eye, and is usually easily called up by a slight external pressure. It has been explained by five or six theories, differing somewhat in detail, but all referring it to the momentary increase of blood tension produced by the contracting heart, which causes either pressure from without on the walls of the retinal veins, rythmically contracting them, or, by a local backward pressure on the venous blood column, rythmically dilating them.

The Diminution in Size of the Retinal Arteries

is due to the fact that the increased intra-ocular tension prevents the normal amount of blood from entering the eye. Von Graefe (31) in 1854, described as a "constant thing in glaucomatous amaurosis," "the presence of pulsation of the central artery of the retina, spontaneous, or produced by light pressure of the finger." To Donders has been ascribed the credit of pointing out that "this arterial pulse can be induced in healthy eyes, also by a gradually increasing pressure on the globe, and that, at the moment when the pulse appears, vision is temporarily abolished" (Snellen, 32); but v. Graefe writes in 1854 (33), "in physiological circumstances one must use a very considerable pressure for the production of the arterial pulse," and Donders (34) himself says, in 1855, "it belongs to v. Graefe to have shown with certainty the appearance of the arterial pulse produced by strong pressure," while v. Graefe, (35), on the other

hand, wrote, in 1857, "through artificial pressure on the globe, we make the arterial pulse to appear (Donders)."

Although a case has been reported by Gowers (36), in which nothing pathological could be discovered to account for it, and which he considered to be always the result of disease, though not necessarily of the eye itself, it is, however, strong presumptive evidence of glaucoma, which is by far its commonest cause. It is due to the altered relationship between the tension inside the intra-ocular vessels, and that outside them. Normally, owing to the small size of the vessels, and to the pressure on their walls exerted by the ocular tension, the stream of blood in these arteries proceeds in an equable flow without visible pulsation, but when the intra-ocular tension is raised to a certain point it causes collapse of the arterial walls except during the heart's systole, when the blood pressure of course is increased, resulting in a rythmical dilatation of the vessel. In a similar way it arises when the intra-ocular tension is high only relatively to that inside the vessel, i. e., when the vascular tension is diminished, as for instance, in the case recorded by Wordsworth (37) where it suddenly appeared in a patient on the point of fainting; in acute anaemia from haemorrhage; and in chlorosis, in which it is due, according to Raehlmann, to hydraemia, and, according to Schmall (38), to "a certain amount of cardiac contraction combined with sudden relaxation of the heart muscles, occurring in certain states of low arterial tension;" in Graves' disease; and in local compression of the central artery within the nerve in optic-neuritis (Fuchs, 39). Retinal pulsation has also been observed when the arterial is relatively greater than the intra-ocular tension, as when the latter is reduced in typhoid fever (Schmall, 40): or, from an increase in the vascular tension in aortic regurgitation (Quincke, 41, Becker, 42, Fitzgerald, 43, S. Mackenzie, 44), in which it is not uncommon; and the greater the hypertrophy of the left ventrical, the more marked it is. It differs from the pulsation of *plus* tension, in that it is often visible, not only on the disc, but well towards the periphery of the retina. "It consists, like the pulsation of other vessels, in a widening and elongation" (Gowers). Berry (46), whilst admitting that the above explanation of glaucomatous arterial pulsation holds good sometimes, asserts, on what grounds he does not state, that, "in many cases, at least, it is due to an active spasmodic contraction of the vessels themselves."

REFERENCES CONTAINED IN CHAPTER I.

1. Fuchs—Lehrbuch der Augenh., p. 376.
2. Brailey—Royal London (Moorfields) Ophthalmic Hospital (R. L. O. H.) Reports, Vol. IX.
3. Priestley Smith—"Glaucoma," London, 1891.
4. Fuchs—Op. Cit. (1), p. 390.
5. Mauthner—Arch. Ophth. N. Y., 1878, p. 178.
6. Fuchs—v. Graefe's Arch. f. Ophth., XXVII, 3, 1882, p. 66, "Ueber Glaukomatoese Hornhauttruebung."
7. Treacher Collins—Ophth. Review, July, 1890.
8. Mauthner—Op. Cit., 5.
9. Dobrowolsky—Arch. f. Ophth., XV., p. 267, quoted by T. Collins
10. Donders—Arch. f. Ophth., VIII., 2, p. 165.
11. Treacher Collins—"Iridescent Vision in Glaucoma," Ophth. Rev., July, 1890.
12. Lauder-Brunton—Lancet, March 3d, 1888.
13. Wurdinger—Klin. Monatsbl. f. Augenh. Ap., 1886, p. 14, quoted by T. Collins.
14. Thomalla—Centralbl. f. Prakt. Augenh., Nov., 1889, quoted by T. Collins.
15. Priestley Smith—Op. Cit. (3), p. 134.
16. Schweigger—Arch. Ophth., XX, 1891, p. 475.
17. Berry—"Diseases of the Eye," p. 197.
18. Fuchs—Op. Cit. 6.
19. Fuchs—Op. Cit. (1), p. 392.
20. Mauthner—Op. Cit. (5).
21. Brailey—R. L. O. H. Reports, X, p. 282.
22. Schnabel—Arch. Ophth. N. Y., VII., 1878, p. 12.
23. Treacher Collins—R. L. O. H. Reports, XIII., 1893, p. 482.
24. Fuchs—Op. Cit. (1), p. 391.
25. Swanzy—"Diseases of the Eye," p. 302.
26. Schweigger—Op. Cit. (16).
27. Knies—Centralbl. f. Allgem. Path. Ap., 1890, p. 249.
28. Treacher Collins—Op Cit. (23), p. 483.
29. Priestley Smith—Op. Cit. (3), p. 131.
30. Lang and Barrett—R. L. O. H. Reports, XII, p. 60.
31. v. Graefe—Arch. f. Ophth., I, 1, p. 372.
32. Snellen—Ophth. Rev., X, 1891, p. 37.
33. v. Graefe—Op. Cit. (31), p. 376.
34. Donders—Arch. f. Ophth., I, 2, p. 81.
35. v. Graefe—Arch. f. Ophth., III, 3, p. 456.
36. Gowers—"Medical Ophthalmoscopy," p. 20.
37. Wordsworth—R. L. O. H. Reports, IV, 3.
38. Schmall—Arch. f. Ophth , XXXIV, 1, p. 37; also (36), p. 19.
39. Fuchs—Op. Cit. (1), p. 18.
40. Schmall—Op. Cit. (38).
41. Quincke—Berlin Klin. Wochensch, 1868, p. 34, and 1870, p. 21.
42. Becker—Arch. f. Ophth., XVIII, p. 206.
43. Fitzgerald—Brit. Med. Jour., Dec. 23, 1871.
44. S. Mackenzie—Med. Times and Gaz,, 1871, 1 (these last four all quoted in 36).
45. Gowers—Op. Cit. (36), p. 19.
46. Berry—Op. Cit. (17), p. 206.

CHAPTER 2.

EXPLANATION OF SYMPTOMS (CONTINUED).

Papillitis.

Most authorities do not include papillitis among the symptoms of early glaucoma, though a condition of hyperaemia and oedema are not uncommonly described, and it is sometimes difficult to know whether or not the authors who mention it consider this a true inflammation which should be classed with the neuritis, which is by some declared to be a usual accompaniment of glaucoma. Thus Jaeger (in 1876) thought the cup was preceded by a true inflammation of the head of the optic nerve, in which he was supported by Klein. Brailey (1) said that when high tension was not of long duration he found microscopically the optic nerve swollen, and the lamina cribrosa yielding backwards; and Mauthner (2) believed the optic nerve to be softened by a morbid process secondary to choroiditis, so as to cup later under normal tension. Brailey and Edmunds (3) concluded from their observations that in primary glaucoma, "Neuritis" precedes the increased tension. Brailey (4) again stated his belief that a pre-glaucomatous stage exists with, among other things, in every case slight haze and swelling of the papilla, and, in the same year he wrote (5) "before plus tension, are always present inflammations of the ciliary body and optic nerve." Gruening (6) recognized a congested disc as one of the early features of certain cases, and present along with the first premonitory symptoms. Knies (7) has written, "among the earliest and most characteristic appearances I found marked hyperaemia and oedema of the entrance of the optic nerve. This appears to be the regular beginning which passes in weeks, into cupping. It fails in no eye which is examined before the cupping;" and Fuchs (8), "the reddening and veiled appearance of the head of the optic nerve during the in-

flammatory attack are produced by hyperaemia with some oedema." Bitzos (9) asserted that "the symptom first seen, constant, and which gives the fundamental picture in primary glaucoma, is 'glaucomatous papillitis.'" It "has a peculiar and characteristic aspect; the papilla becomes soft, slightly oedematous, reddish yellow and then grayish yellow, which gives it a resemblance to the papillitis of retinitis pigmentosa. The arteries are already contracted and the veins dilated. The inflammatory process reaches, to some extent "the peripapillary choroid. The papilla soon begins to cup, the vessels form little curves" etc. This papillitis is seen, according to this author, by chance when examining healthy eyes, or when the other eye is already glaucomatous. The vision and the visual field are both subnormal, there is no increase of tension if the stage of cupping has not begun, which takes place about two months after the beginning of the papillitis. Then the oedema diminishes, the papilla becomes grayer, and *plus* tension appears. He says, "it seems to me that one can accept as an absolutely certain fact that the glaucoma has begun by a papillitis constituting a lesion which is primary and at the same time fundamental"; and, "we do not meet with commencing glaucoma without a papillitis, more or less marked when seen from the beginning. Further the glaucoma begins by a papillitis on one side when that on the other is already advanced. Besides, the glaucomas, which begin under our eyes in the form of papillitis are the most distinct and incontestable; finally, pathological anatomy proves to us that optic-neuritis is a very early lesion of glaucoma, and that it forms the only constant lesion." Zentmayer and Campbell Posey (10), in discussing 167 cases of simple glaucoma found in the books of the clinics of Drs. Norris and Oliver at the Wills Eye Hospital, Philadelphia, conclude that "an inflammation of the optic nerve is a constant attendant upon glaucoma, being noted in every eye containing a pathologic excavation. It manifests itself as a low-grade neuritis, affecting the entire structure of the nerve, and seems to render the nervous tissue more liable to the peculiar kind of excavation which is the most constant characteristic of glaucoma." These words may be mistaken as meaning that these authors have ground for the assertion that the other glaucomatous symptoms follow, and possibly even result from, optic neuritis; but, from a careful consideration of their previous

statements, no such conclusion appears tenable, and all that can be fairly deduced is that along with, or as the result of, other glaucomatous symptoms the optic nerve also in glaucoma is abnormal. This subject has an important bearing on the question of the etiology of glaucoma, in which section it will come up again for consideration.

Cupped Disc.

It is usual to describe three forms of cupping of the optic disc, which it is important to distinguish from one another. These are called physiological, atrophic, and glaucomatous cups.

The Phsysiological Cup

is exceedingly common, and is the result of the early separation, after passing through the lamina cribrosa, of the fibres of the nerve on their way to divide up for the formation of the retina. It is therefore shaped rather like a flower vase than a cup, narrow below and opening up widely at the mouth, and the retinal vessels can be followed all the way down its sloping walls. It occupies only a portion of the disc not usually central, but more often to the temporal side. It is paler than the remainder of the disc. On section the lamina cribrosa is found to be at its proper level. These cups are always bi-lateral.

The Atrophic Cup

is formed by the disappearance of more or less of the nerve structure in front of the lamina cribrosa, which retains its normal level, and limits the depth of the depression. It is shaped like a shallow saucer, modified, of course, by the original physiological form of the disc, and the vessels are visible during their entire course upon the disc. The whole of the disc is usually affected, and the perforations of the lamina cribrosa are often unnaturally distinct.

The Glaucomatous Cup

differs from each of the preceding, chiefly on account of the depressed level of the fenestrated membrane, which fills up the optic foramen in the sclera, and through which the nerve passes—the lamina cribrosa. Its depth is therefore greater; and, moreover, in cases of some duration the sides are under-

mined so that the vessels after curving sharply over the edges of the disc disappear from view only to become visible again at the bottom of the cup. It appears as if the high intra-ocular pressure had excavated the head of the nerve as far as possible in every direction. The lamina cribrosa being delicate early yields before it, and the undermining of the sides, a later stage,

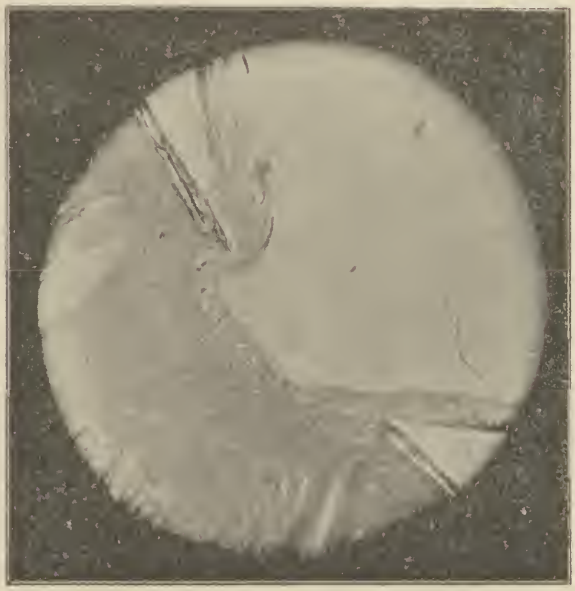

FIG. 2.—GLAUCOMA CUP. The nerve tissue is atrophied, the lamina cribrosa driven back, and the side excavated. *

is facilitated by the fact that the scleral foramen through which the nerve passes is wider behind than it is in front. The nerve tissue has first a full color, becomes later pale and atrophic, and the vessels are pressed hard against the lips and the walls of the cup, leaving ultimately only a narrow rim to represent what had been the normal position of the disc. In advanced cases the visible vessels are found to be congregated chiefly at the side. The bottom of the cup is not to be seen distinctly with the lens suitable for the level of the retina, a lower plus or higher *minus* being required, and its depth may be measured by allowing 3 dioptres for each millimeter of difference. The inequality of depth can be proved also by noting the parallactic movements which occur when the observer, using the direct

*Most of the illustrations in this work were first photographed from sections of eyes removed in Moorfield's Hospital, London, and prepared in the laboratory there under the supervision of Mr. Treacher Collins.

method, changes the point from which he views the fundus; or, with the indirect method, when he moves his object lens.

In late stages, as already said, the cup has been observed to fill up again by the formation of new tissue in it (Brailey, Pagenstecher, etc.), and it has also been seen to be completely filled with blood (Hartridge, 11; Morton, 12).

It should be noted that occasionally the disc is cupped in a manner "almost typical" of glaucoma, "with normal vision and visual field, and without another symptom of glaucoma;" but Nettleship (13) says of these cases, "we cannot but be very careful in inferring that a person whose discs show changes resembling those of glaucoma, is really safe, however, free he may be from the symptoms and other signs of the disease."

Mackenzie (14) seems to have been the first to note the change in level of the optic disc in glaucoma, for he said, in 1854, "In this, as well as in another case which I had an opportunity of examining after death, the optic nerves were flattened and atrophic." In the same year, Jaeger (15) noted the peculiarity of the disc, with the ophthalmoscope, and he, and v. Graefe also, expecting to find signs of inflammation, mistook it for a swelling, and the latter (16) said of it, "the change in the opticus, which has already been carefully studied and illustrated by several observers, especially by Ed. Jaeger, consists in that the optic nerve entrance shows in nearly its complete circumference a strongly marked round elevation, and only a thin peripheral zone lies at the natural level of the retina which borders closely on the elevation like a ring laid round its base." The following year Adolph Weber (17) demonstrated how such mistakes might be avoided by noting parallactic movements on the fundus, and the lenses required to focus the different depths. Desmarres (18) wrote in 1858, "One thought at first that the papilla of the optic nerve became convex in glaucoma. Since then one has recognized that, on the contrary, it is concave." In the same year Mueller (19) repeated on an excised eye the observation made four years earlier by Mackenzie, and said, the disc "forms a concavity from whose border the retina rises up perpendicularly. The walls of the pit sink from above through the normal choroidal ring, at first very steeply, so much so that in many places the border somewhat overhangs." The symptom thus brought to the notice of the profession has since by most ophthalmologists been ascribed to the simple mechanical pressure of the intraocular tissue pushing back the lamina cribrosa, which is the weakest part of the globe, and at the same time, causing atrophy and sclerosis (Brailey and Edmunds, 20) of the nerve fibres, here entering the eye, which still farther enlarges the excavation. Von Graefe (21) in 1862 asserted that "every increase of tension,

influencing the conductibility of the retina, leads also to a *characteristic* excavation of the optic nerve." In 1875 Schmidt Rimpler remarked that the cup was "characteristically glaucomatous when the lamina cribrosa had been pushed back." Schoeler (22) in 1879, by cauterizing the corneo-scleral limbus, produced T+2 or T+3, which quickly caused the papilla to blanch, the arteries and veins becoming almost invisible throughout or only at their central ends, and running to the periphery as fine pale red lines; the more marked the increase of tension the less are the vessels visible." The short-lived tension produced by these experiments Schoeler asserts to have been sometimes sufficient to excavate the disc.

Priestley Smith (23) says that "there is ample evidence that the typical excavation of the papilla—the glaucoma cup—is a product of high pressure. It is to be found, by dissection or otherwise, in every eye which has suffered long or frequently from high tension. It is not to be found after a very brief period of high tension, for the excavation involves atrophic changes which need time for their development, but even in such cases the microscope shows the first step of the process—a pushing back of the lamina cribrosa; and even in the healthy excised eye high pressure artificially applied causes the same displacement," and again (23) "it is not a purely mechanical result of pressure; it includes atrophic changes, which, though essentially due to pressure take time for their development;" also, "when the excavation of a disc proceeds very slowly the damage done to the nerve fibres and the corresponding impairment of the field are sometimes very slight as compared with the depth of the cup" (23). "It is possible of course that a similar excavation may arise from causes other than an excess of pressure. Should this prove to be the case, it will be well to give such cases some name other than glaucoma."

Treacher Collins (24) wrote, "The seat of entrance of the optic nerve is a weak point in the walls of the eye, and the lamina cribrosa early becomes depressed backwards. This depression backwards of the lamina cribrosa, together with atrophy of the nerve fibres, causes cupping of the optic nerve." These words express the general opinion of ophthalmic pathologists, and they are supported by a consideration of the fact that cupping is found in cases of secondary glaucoma distinctly due to some fault in the anterior part of the eye, as from dislocation of lens (Treacher Collins 24), from serous iritis, or in buphthalmia from hereditary malformation of the anterior chamber. It will be observed, that among others, the authors just quoted, make no mention of a morbid condition of the optic nerve antecedent to the pressure, but that they imply that any changes in the disc, peculiar to the glaucoma process, are secondary to the increase of intra-ocular tension.

On the other hand, cases have been reported in which there was cupping but no *plus* tension, others in which there was long continued high tension without cupping; and still others in which the high tension was known to have been preceded by a morbid state of the papilla considered to have been of more importance in the production of the cupping than was high tension, which, however, always followed. In connection with this last class, we have already seen that Jaeger, Klein, Brailey, Edmunds, Mauthner and Bitzos have distinctly stated their opinion that a morbid process attacks the nerve head before the appearance of plus tension; whilst, among others, Gruening, Knies, and Fuchs consider that an unnatural condition of the disc is one of the earliest symptoms of glaucoma, though I cannot include them among those who hold that the appearance is actually anterior to the increase of tension. Mauthner farther wrote: "At the beginning of the excavation pressure is often not raised." . . . "A morbid process attacks and softens the lamina cribrosa and optic nerve so that the lamina yields to normal pressure; the supporting tissue of the optic nerve gives way, the fibres of the optic nerve become extraordinarily transparent, and on the other hand the vessels sink backwards into the transparent head of the optic nerve. Thus it seems as if the latter were pressed close to the scleral ring while the masses of nerve fibres covering them cannot be seen directly with the mirror." He supported this theory by stating that he had found transparent matter in the cup after excision of the eye. The atrophy, he remarked, was undoubtedly increased by heightened tension. The peculiar convoluted vessels sometimes seen at the bottom of the cup were new formations in connection with the morbid process. Mauthner admitted that the explanation was very forced, and said that he would welcome a better.

Bitzos (26) conceded that, as ordinary "optic neuritis does not render the lamina cribrosa more feeble or produce *plus* tension, we must suppose something peculiar about glaucomatous optic neuritis, yet the disc begins to cup and the vessels to curve without doubt before plus tension appears, but these things become more marked on account of *plus* tension."

Glaucomatous Excavation without Increased Tension.

It has long been known that typical glaucomatous cups may exist without any apparent increase in intra-ocular tension; for example, Schmidt-Rimpler (27) mentioned this in 1875, and stated that the optic nerve fibres, at first normal, later become atrophic. Schnabel (28) reported a case* of glaucoma seen in

*It may be observed in connection with this case, that previous high tension might have so weakened the lamina cribrosa as to make it unable to resist a normal amount of pressure

an early stage and without cupping, on which he performed iridectomy, with the result that there was no longer plus tension, and yet a typical cup afterwards formed without, however, any further reduction of vision. Nettleship (29) said "the tension in the quietest forms is sometimes never perceptibly increased," and "the depth of the excavation bears no constant relation either to the atrophic pallor of the disc or to the degree of tension of the eye." Stedman Bull (30) in 90 cases of simple glaucoma, found in both eyes normal tension in only two cases. Knies (31) said, "the cup is known to appear without plus tension, and with quite good vision." Nettleship (32) had already discussed the relationship existing between glaucoma and excavations appearing in eyes in which there had previously existed large physiological cups, and expressed himself with considerable caution on the subject, but Schweigger (33), at a later date, in connection with these cases, asserted his absolute belief that they are not glaucomatous at all, but are due to atrophy; in glaucoma there must always be perceptible increase of tension. (For further discussion of this topic, see Diagnosis.) Chauvel (34) expressed a very commonly accepted opinion when he said, "One ought to look upon the *plus* tension as a necessary condition, only in chronic cases it is hard to feel it, as the eye gradually fits itself to the altered pressure." Priestley Smith (35) maintained that the hypothesis is unnecessary which assumes that glaucoma "is the expression of some unknown agent which usually raises the pressure and excavates the disc at the same time, but which occasionally excavates the disc without raising the pressure." And, he continued, "it is very probable, therefore, that these cases of glaucoma with normal tension are cases which have been examined only during the intermissions.

Increased Tension Without Cupping.

A few cases have been recorded in which, after even a very considerable time, there was no cupping. The length of time necessary for the formation of a pressure cup is given very variously by different authors. Mauthner (36), for instance, went so far as to assert that glaucoma may become "absolute" without cupping, and he had seen cases of chronic inflammatory and of simple glaucoma which after a year's duration showed no cup; but he gave no account of any microscopic examination of the condition of the lamina cribrosa. In contra-distinction to this Schoeler has found cupping in rabbits' eyes after an artificially produced T+2 or 3 of some two hours' duration. We have seen that certain writers believe the cupping to precede the *plus* tension; and Bitzos (37) asserted that the cupping begins about two months after the first appear-

ance of papillitis. In glaucoma fulminans the eye may be blinded in a few hours or less, and without cupping, and operation may cure high tension while leaving the eye blind and without cupping, as in a case of Mauthner's of six days' duration, and one of Rydel's of three weeks' duration. Brailey (38) reported a case of very high tension for nine days in which no cup was found microscopically. He had previously reported a case of six weeks' duration, T + 3, at the time of the excision, where there was a pitting of the nerve, but not yet any lateral excavation, and another, T + 2, at the time of the excision, of five months' duration, with a wide and shallow cup, but no lateral excavation. "I find no case with a cup whose edges are distinctly undermined without a history of at least two years, and in many cases with a history of seven or more years, and T+1 at excision, the cup is neither deep nor undermined laterally." Knies asserted that it takes weeks for the hyperaemia of the disc to pass into cupping, and Schweigger (40), "as far as my observation goes the excavation of a level disc proceeds for at least six months before it actually reaches the papillary border." Priestley Smith (41) wrote, "cupping of the disc is not to be discovered during or after a first attack of acute glaucoma, unless the outbreak has been preceded by a chronic excess of pressure of some months' duration," but depression of the lamina cribrosa may be found. Treacher Collins' (42) report on the glaucomatous eye in the Moorfields hospital museum, Case VIII, is described as exhibiting cupping after a raised tension of five months' duration; and Case XVII, one of haemorrhagic glaucoma, after two and a half months V. Garnier (43), reported a case of *plus* tension of six weeks' duration occurring after a traumatic cataract caused by a powder explosion, in which examination showed a deep cup up to the margin of the disc.

One is safe to conclude from the above examples of what has been written on the subject, that cupping of the disc is an invariable result of the glaucomatous process when that has existed for a certain, though indefinite, time, the exceptional cases being, as Priestley Smith remarks, those in which the disc has been protected by a new growth.

The Halo Glaucomatosus.

A white circle surrounding the disc is usually held to be due to atrophy of the choroid, the result of pressure; and occasionally the retina, choroid, and sclera have been found united in that region by adhesions (Knies). Mauthner considered this to be part of the process affecting choroid and disc which, in his opinion, was the cause of the excavation, the choroidal atrophy following an exudation under it. When it appears early it is probably as a result of such exudation.

Pain

in the eye and head is the result of pressure on the ciliary branches of the 5th nerve, especially in the ciliary body and iris, the sensation being transferred from the actual seat of the disease to other regions supplied by the nerve, and especially by its first and second branches. It seems reasonable to suppose that the dragging on the zonule, due to the forward pressure of the lens, and the nipping of the iris and ciliary processes between the lens and sclera, may be a common cause of pain.

The Frequently Rapid Advance of Presbyopia

is said to result from pressure interference with the action of the third nerve on the ciliary muscle; to a tension of the choroid from stretching, due to the internal pressure, which interferes with the power of the ciliary muscle in drawing it forward for the purpose of accommodation (Priestley Smith); to a diminution of the refraction from a nearer approach of the eye to a globular shape (Swanzy, 46); while the rapid atrophy of the ciliary muscle, said by Brailey to be an early result of the glaucoma process, might have been brought forward to account for this symptom.

The Photopsia,

which is sometimes complained of even when the patient is quite blind, is in all probability the result of violent stimulation of visual nerve fibres, caused by a stretching or dragging of the retina.

The Disorganization,

which follows upon glaucoma absolutum, consists in a degeneration of the various tissues, with an alteration in the shape of the globe, which tends to become square from bulging between the recti. The cornea is now likely to show one or other of the following appearances: Bullae, due to the raising up of the epithelium by serum below it; superficial opacity of the whole cornea, along with one more deeply seated and limited in extent; ulceration, or abscess, perhaps with perforation; "transverse films." The removal of epithelium, which occurs so easily when it is elevated by exudation beneath, facilitates the entrance of infective material, which may result finally in panophthalmitis. The lens becomes cataractous and often calcareous, and a band of hyaline globules has been found to cross its nucleus (Treacher Collins). The atrophy in the ciliary region causes the vitreous to shrink and become detached from the retina. Degenerated vessels in the retina give rise, not infrequently, to haemorrhages into its substance, and occasionally it becomes detached from the choroid

from one or other of various causes—the previous shrinkage of the vitreous; haemorrhages, from changes in the vascular walls rendering them brittle; dragging of organizing inflammatory products; or, as Knies suggested, shrinking of the membrana limitans interna. The anterior part of the retina is frequently cystic.

The Deterioration of Vision.

Temporary deterioration in visual acuity is no doubt caused by the hazy condition of the cornea, and possibly of the aqueous, together with the interference with the retinal nervous functions, probably the result of choroidal oedema, due to intraocular pressure. Schnabel thought the reduction of vision to result from spasmodic contraction of retinal vessels. When the visual nerve fibres have become organically deteriorated, the resulting interference with sight is permanent. Along with diminished acuity of central vision is found a contraction of the visual field which was first observed by Mackenzie, but first studied by Hoffmans under the guidance of Donders, the results being published in Dutch in 1860 (Snellen, 48). In acute cases this contraction is comparatively not so apparent as is the general haziness of vision affecting the retina as a whole; but in those which are more chronic, and in which the tissues gradually accustom themselves to new conditions, even to considerable excavation of the disc, the central vision may remain fairly good, while parts of the periphery are more or less blind. Most frequently it is the nasal side which earliest and chiefly suffers, but this is by no means constant. Thus, Bunge (49) found in 100 cases a defect in the nasal side alone in 27; predominating in the nasal portion in 44; field remaining in the form of an oval around the papilla in 4; in 9 cases the whole field was destroyed, center included, except a small portion on the temporal side, a central or paracentral scotoma, with or without some nasal contraction in 4; contraction upwards alone, in 2 cases; concentric restriction, in 6; restriction, chiefly in temporal side, in 2 cases. The contraction of the field for colors starting each from its own normal, proceeds as a rule *pari passu* with that for white, or at most a little faster, which is worthy of note as a point of difference between glaucoma and optic atrophy. The precise cause of the glaucomatous restriction of the field is not yet definitely settled. It is probably due either to the fibres in the optic nerve destined for the peripheral zone of the retina being more exposed to damage at their entrance into the eye (the central nerve fibres, according to Bunge, though Donders thought that the nerve fibres for the periphery lay most superficial in the nerve trunk, and were therefore most exposed) or, because the more distal vessels are smaller and more susceptible to pressure, and especially on the

temporal side where the vascular supply is weakest; or, because oedema affecting the retina would naturally be most marked at the greatest distance from the vascular centre. It is evident, in any case, from the variety in the forms of the field, either that the anatomical relationships of the tissues involved are inconstant, or that the cause is not always one and the same.

REFERENCES CONTAINED IN CHAPTER II.

1. Brailey—R. L. O. H. Reports, IX, 1877.
2. Mauthner—Arch. Ophth. N. Y., VII., 1878, p. 178.
3. Brailey and Edmunds—R. L. O. H. Reports, X, 1880, p. 86.
4. Brailey—R. L. O. H. Reports, X, 1881, p. 275
5. Brailey—R. L. O. H. Reports, X, p. 282
6. Gruening—Trans. Am. Ophth. Soc., 1889.
7. Knies—Centralbl. f. Allgem. Path., April, 1890.
8. Fuchs—Lehrbuch der Augenh.. p. 392.
9. Bitzos—Ann. d'Ocul., CXII, 1894, p. 92.
10. Zentmeyer and Posey—Arch. Ophth. N. Y., 1895, XXIV, p. 391.
11. Hartridge—Trans. Brit. Ophth. Soc., X, 1890, p. 64.
12. Morton—Trans. Brit. Ophth. Soc.. X, 1890, p. 155.
13. Nettleship—R. L. O. H. Reports, XII, 1889, p. 72.
14. Mackenzie—"Diseases of the Eye," 4th Ed., 1854, p. 897.
15. Jaeger—"Ueber Staar und Staaroperationen," Vienna, 1854.
16. v. Graefe—Arch. f. Ophth.. I, 1854, p. 371.
17. Weber—Arch. f. Ophth., II, 1855, p. 141.
18. Desmarres—"Traité Théorique et Pratique des Maladies des Yeux," III. p. 733.
19. Mueller—Arch. f. Ophth., IV, p. 77.
20. Brailey and Edmunds—Op. Cit. (3).
21. v. Graefe—Arch. f. Ophth., VIII, 2, p. 280.
22. Schoeler—Arch. f. Ophth., XXV, 4.
23. Priestley Smith—"Glaucoma," pp. 3, 137 and 4.
24. Treacher Collins—R. L. O. H. Reports, XIII, 1893, pp. 484 and 495.
25. Mauthner—Op. Cit. (2).
26. Bitzos—Op. Cit. (9).
27. Schmidt-Rimpler-Graefe- Saemisch Handbuch.
28. Schnabel—Arch. f. Augenh., V, 1876. p. 50.
29. Nettleship—R. L. O. H. Reports, XII, 1889, p. 72.
30. Stedman Bull—Trans. Am. Med. Soc , 1889.
31. Knies—Op. Cit. (7), p. 249.
32. Nettleship—Op. Cit. (29.)
33. Schweigger—Arch. Ophth. N. Y., 1891. XX, p. 475.
34. Chauvel—Bull. de l'Acad. de Med. de Paris, XXXI, 1894, p. 240, for Galezowski.
35. Priestley Smith—Op. Cit. (23), p. 2.
36. Mauthner—Op. Cit. (2).
37. Bitzos—Op. Cit. (9).
38. Brailey—R. L. O. H. Rep., X, p. 258.
39. Knies—Op. Cit (7).
40. Schweigger—Op. Cit. (33).
41. Priestley Smith—Op. Cit. (23), p. 137.
42. Treacher Collins—R. L. O. H. Reports. XIII, pp. 488, 501.
43. v. Garnier, Arch. f. Augenh., XXV, 1892, p. 24.
44. Knies—Op. Cit. (7).
45. Priestley Smith—Op. Cit. (23), p. 135.
46. Swanzy—"Diseases of the Eye," p. 302.
47. Treacher Collins—Op. Cit. (42), p. 484.
48. Snellen—Ophth. Rev., X, 1891, p. 39.
49. Bunge—"Gesichtsfeld," 1884.

CHAPTER III.

ETIOLOGY.—GENERAL CONSIDERATIONS.

Until Helmholz, in 1851, armed the profession with the ophthalmoscope, it was working, in the matter of glaucoma, literally in the dark. Then a new interest was given to the subject, which was materially enhanced five years later, when Graefe showed by his use of iridectomy that this previously practically incurable disease was susceptible of beneficial treatment.

Physicians in the olden times were acquainted with the later stages of glaucoma, but it was only comparatively recently that the differential diagnosis between it and other diseases, especially cataract, was placed upon anything like a sure foundation.

Hippocrates comprehended under the term glaucoma every sort of opacity which appears behind the pupil, but he distinguished varieties of these without knowing in what the differences consisted. The later Greeks called incurable opacities γλαυκωαα while the more favorable they named ὑπσχαμά the former depending on changes in the lens, and the latter on an accumulation between the iris and the lens. "Abundant proof could be brought that these were the doctrines of Galen in 1538, and of Rufus in 1557" (Mackenzie, 1), and that no advance had been made down to the time of Brisseau, in 1709. "Even Maitre-Jan (in 1711), to whom we are, in a great measure, indebted for establishing by dissection the fact that cataract is, in general, an opacity of the crystalline lens, and not a filmy substance between that body and the iris, still maintained that glaucoma was also a disease of the lens, "a peculiar alteration in the lens, by which it dries up, diminishes in volume, changes its color, and loses its transparency, whilst preserving its natural shape, and becoming more solid than it ought to be naturally." (Mackenzie, 1) Maréchal's examination of the eyes of Dr. Bourdelot, physician to Louis XIV, and the fact that couching did not relieve the condition, were the foundations for Brisseau's assertion in 1709 that glaucoma is a disease of the vitreous such as cataract is of the lens. Boerhaave, (2) in his treatise on Diseases of the Eye, published in Paris in 1748, still included glaucoma under the heading of cataracts. "Pains, great, profound, pertinacious and of long duration, at the fundus of the eye, produce sometimes a cataract; then, finally, the brilliance of the eye disappears and an opaque appearance takes its place, when the cataract is fully developed. . . . If the humor collected there changes its color into yellow, green, black, etc., it will be a spurious cataract, but such cataracts were never willingly touched by any of the ancients. . . . Hence arise two separate conditions,—in some persons the iris remains mobile with vision; in others it becomes immobile with blindness." Later,

the idea, quoted by Beer, who also considered the disease arthritic in nature, that glaucoma is a disease of the vitreous, gained ground; Wardrop, for instance, writing in 1818, that the "vitreous humor acquired a dull, greenish color, accompanied with insensibility of the retina, a species of amaurosis." Prof. Rosas, of Vienna, in 1834, described three varieties, glaucoma of the hyaloid, of the retina, and of the choroid, and Mackenzie asserted in 1854 that the general idea still remained that glaucoma is a disease of the vitreous. Mackenzie, himself, however, held otherwise, and described it as an inflammation of the choroid from whose internal surface effusion takes place, the retina being compressed, and the vitreous disorganized and replaced by superabundant watery secretion. "It is probable that the aqueous fluid which fills the place of the vitreous humor, becoming superabundant, promotes by pressure the absorption of the pigmentum nigrum, and completes the insensibility of the already disordered retina."

Mackenzie may be said to be the link which connects the days of ignorance and guess-work in the pathology of glaucoma with the modern days of scientific research. He was evidently the first to observe the glaucoma cup, while as early as 1830 he had drawn attention to the hardness of the globe and its importance, and on that basis he performed the operations of sclerotomy, and of paracentesis of the cornea, although usually with only temporary success. But how far he had advanced is seen when we remember that his idea of the choroidal origin of glaucoma, though still mingled with some old-time notions, was closely allied to what has been held down to the present time by many eminent authorities possessed of modern microscopes, brought up to the use of the ophthalmoscope, and in full knowledge of iridectomy; while the common accident of the prolapse of a piece of iris during his operation of opening the anterior chamber, with its subsequent excision, might have antedated the discovery of the best operative measures by over a quarter of a century. After the new methods had given new impetus to the study of glaucoma, novel ideas began to spring up in all directions, and many theories have since then been advanced to explain the symptoms of the disease, a short study of which is necessary in order to understand the present position of our knowledge of the subject.

It is held by the majority of ophthalmic surgeons that the increase of intra-ocular pressure found in glaucoma is responsible for all the evils which accrue to the eye from the disease; in other words, that the conditions giving rise to the high tension, and without the addition of the tension, would have comparatively slight results. Mackenzie (1), as already said, first observed it, appreciated its importance, and brought

it before the notice of the profession. Bowman, in his lectures at the Ophthalmic Hospital, Moorfields, in 1847, made special note of the tension, and at the meeting of the British Medical Society in London, in 1862, he proposed certain formulae for recording its amount, which have since been generally adopted in the somewhat simplified form in which he had them afterwards printed (3); thus, T. n. means a normal tension; T.+3, stony hardness, and T.+1, and T.+2, the degrees which intervene. T-3 is a perfectly soft and flabby eye, and T-1 and T-2 are intervening degrees of softness, while T.+? and T-? mean conditions of uncertain hardness or softness. These have proved to be very valuable formulae, but it is evident that they are not exact, and differences of opinion are frequent concerning the same eye, due, generally, to the imperfection of the method, but at times, no doubt, to the fact that palpation of a hard eye not uncommonly has the effect of reducing the tension. Neither is there any definite standard of normal pressure, which, though the same for the two eyes of any individual, varies to some extent in different persons. Bowman's method, and the one generally in use, is, with a finger of each hand, the patient looking down, to palpate the eye through the upper lid, pressing alternately with each finger. This has the disadvantage that a varying thickness of lid is included in the examination, and to avoid it, Coccius (4) pulled down the lower lid and placed his fingers directly on the sclera. This is more uncomfortable for the patient, but is preferred by a few surgeons, Schweigger (5) among others. In order to get results more scientifically correct various inventors have constructed machines, but no one of them has yet come into general use. Among these are the "Tonometers" of Priestley Smith, Fick (6), Nicati, and Koster (7). The principles to be observed in constructing a tonometer have been set down by Priestley Smith (8). He adds, that no tonometer, "however ingeniously devised, can accurately measure the intra-ocular pressure, or accurately compare one eye with another," but the instrument which he has devised is useful in determining the changes which take place in one and the same eye, and also in comparing the two eyes of the same patient, for which purpose he habitually employs it.

The normal tension, measured by the manometer, is about 25 m. m. of mercury, and is practically as great in the aqueous as in the vitreous chamber (Schoeler, 9, and Priestley Smith, 8). It is maintained by the intrinsic power of the eye automatically to regulate the amount of its fluid contents. In connection with this subject, it may be well briefly to describe the apparatus through which the ocular fluid circulates and the manner in which it acts. The eye is composed of a slightly elastic

frame-work, tough, strong and dense, for the protection of its delicate contents, and to maintain its shape, the anterior part of which, the cornea, is transparent to permit the rays of light to reach the fundus. The sclera is lined by the first part of the uvea, the richly vascular choroid, expanding forward behind the level of the corneo-scleral junction into the second part of the uvea, the outer portion of which, formed by radial and circular muscular fibres is the ciliary muscle, and the inner, the ciliary body, is composed chiefly of blood-vessels. From the ciliary body projects the third part of the uvea, the iris or curtain of the eye, which by reflex action regulates the amount of light which may enter its interior. Its tissue, besides a pupillary sphincter muscle, and (probably) radial muscular fibres as well, contains blood-vessels running mainly in the direction of the pupil, and is backed by a double layer of pigment which cuts off the light from entering the vitreous through its substance. Somewhat internal to the posterior pole of the globe, the optic nerve passes through a foramen in the sclera and the choroid to break up and be spread out fine in a modified form, as the retina, on the internal surface of the uvea. The cavity of the sphere is unequally divided into two by a diaphragm formed of the lens, slung centrally behind the pupil by numerous bands, the suspensory or zonular ligament, passing tightly from its entire circumference to the anterior ending of the choroid proper; the posterior portion containing the semi-gelatinous vitreous, and the anterior the aqueous humor, which communicate through the open meshes of the ligament.

The uvea, the great vascular membrane of the eye, receives its blood supply from branches of the ophthalmic division of the internal carotid artery which pierce the sclera near the optic nerve, and immediately break up in the choroid, therefore "short" ciliary branches, the "long" ones running forwards between choroid and sclera entire towards the iris before dividing, when they are joined by the anterior continuation of the short vessels, and by "anterior ciliary" vessels which enter the eye in front of the insertion of the recti muscles. From a main circular vessel, thus formed, the iris is supplied by branches converging towards the pupil. The blood from all these is returned by from four to six "vortex veins" formed by the junction in the choroid of the smaller vessels, and piercing the sclera in an opaque direction backward, some thirteen m. m. behind the cornea, generally close to, but not under, the straight muscles (A. W. Stirling, 10), except a small quantity carried off by the anterior ciliary veins, which like their corresponding arteries, pass through the sclera not far behind the cornea. The central artery of the retina, entering the eye upon the disc, does not afterwards form any anastomosis among its own branches or

with the vessels of the choroid, but with the latter it does form a small anastomosis round the optic nerve close to the eye. The fluids of the interior of the eye are believed to enter at the ciliary body, (perhaps a small quantity enters through the sheath of the optic nerve) to circulate by passing between iris and lens, and to leave at the angle of the anterior chamber, a small proportion only escaping around the central vessels of the optic nerve. The following are grounds for this belief: Besides the difficulty of seeing any other likely source for the fluid, what other sufficiently important uses the highly vascular and convoluted ciliary region could subserve, the suggestive nature of its epithelial covering, and the firm attachment here of the vitreous, numerous experiments have been made which generally add strong support to the idea that this is the great secreting portion of the eye. As well as the recent writings of Treacher Collins, who claims to have found the secreting glands, of Nicati, of Greef, and of John Griffith who actually saw beads of secretion, there are the older experiments of Deutschmann (11), who, after removal of the ciliary processes and iris in rabbits, found that the vitreous and lens became destroyed, and that the aqueous ceased to be formed; and of Schoeler along with Uhthoff (12), who observed that after subcutaneous injections of fluorescin the coloring matter passes from the ciliary body, and perhaps a little from the back of the iris, rapidly into the aqueous and more slowly into the vitreous. Leplat (13) injected potassium iodide subcutaneously, removed the eyes, and testing quantitatively for the salt, confirmed the researches of the others. The manner of its exit is easily understood. Ulrich (14) asserted that it passed through the iris, which on the face of it is unlikely, and is disproved by the fact that when the periphery of the iris adheres all round to the lens capsule the iris is bulged forward by the fluid behind it. That it escapes at the angle of the anterior chamber, and by filtration, was shown by Leber who found diffusible coloring matter passing out by Schlemm's canal and the iritic veins, while colloid material did not do so. The cornea was impermeable even to diffusible bodies, when the layer of epithelium on its posterior surface was in normal condition. Priestley Smith (15) undertook a number of interesting experiments in which he passed fluid into the vitreous and aqueous of newly excised eyes by means of canulae. The result pointed to the following conclusions: (1) that fluid injected into the vitreous chamber escapes chiefly if not entirely through the aqueous chamber; (2) that a slight excess of pressure in the vitreous chamber displaces the lens and iris forwards, compresses the filtration angle and impedes the escape of fluid; (3) that there is no escape through the vortex veins when the filtration angle is compressed; (4) that there is

little if any escape at the papilla. Leplat (16) having later, by experiments on living rabbits' eyes, shown that an increase of pressure in the vitreous does not displace the lens forward, Priestley Smith explained the discrepancy between these results and his own by stating that in the living eye it is probable that a rise in pressure in the vitreous is immediately followed by a similar rise in the aqueous. Leplat agreed in finding the escape of fluid in the optic nerve to be very small indeed, though after his earlier experiments with potassium iodide he had concluded that the papilla contained an important channel for this purpose, and others have found reason to support the latter view. Schwalbe's (17) lymph spaces in the optic nerve sheath were believed by him to carry off lymph from retina, vitreous and optic nerve. Gifford (18) injected fluid containing insoluble colored particles into the vitreous, which he saw with the ophthalmoscope, collected at the papilla, and on section the microscope showed the grains passing backward along the lymph spaces *surrounding the central vessels* on their way to the sphenoidal fissure. But though Stilling (19) saw fluid escape from the nerve under pressure, Priestley Smith (20) could find by experiment with colored fluid, neither that it left the eye at the nerve, nor when injected into the sheath could it be made to enter the eye; while Schoeler (21) and Uhthoff saw no sign of fluorescine leaving the eye in that region; nor in previous experiments could Schoeler find that obstruction there lessens the amount of that fluid. Knies (22) has recently supplemented his previous experiments with ferro-cyanide of potassium (23), and holds that the colored diagrams of his results confirm his old contention that the solution passes into the cornea through Descemet's membrane, and justify the assertion, that even in normal circumstances, part of the aqueous leaves the anterior chamber in this way.

In the first stage, the solution injected into the vitreous is found to leave a blue stain, in breadth one-quarter the diameter of the sclera, passing obliquely backwards through the latter to a point behind the equator where it opens into the lymph space of Tenon's capsule. In the second, a colored streak passes forwards, involving the posterior part of the cornea and Descemet's membrane, and this is supposed to prove that it has penetrated from behind and through Descemet's membrane and its endothelium. That appears to be a quite unnecessary assumption, for it is continuous with the scleral streak, and may just as likely pass into the cornea at the angle of the chamber, which in all the diagrams is wide open; and, even if that were closed by albuminous or cellular exudation, the color which had yet been able to penetrate the sclera might just as well have penetrated the cornea from the same point.

At this stage the color is all at the back of the cornea, not unlikely because it enters from the angle at the back, and because the cornea is composed of stratified laminae. In the next stage, it has occupied the whole cornea, and is making an exit through the sub-conjunctival tissue; while in the last, the posterior part of the cornea is clear, and the color is confined to the anterior layer, and the sub-conjunctival tissue. The solution, whatever be its means of entrance, evidently leaves the cornea by passing gradually forward through its lamellae to the front, and then into the loose tissue under the conjunctiva. A small quantity of fluid, Knies considers, leaves the eye at the optic nerve.

We may reasonably conclude, from all that has been said, that the bulk of the fluid leaves the eye at the angle of the anterior chamber and that probably a little escapes around the central vessels of the optic nerve, but not through the nerve sheath, which more probably transmits it towards the vitreous.

By a consideration of this sketch of what is material in the anatomy and physiology of the eye, one at once observes the close relationship which must exist between abnormalities in the entrances and exits for the ocular fluid and the question of tension. Except under extraordinary circumstances the tissues liable to a change of volume are the fluids, and, by a species of automatic arrangement, these in ordinary conditions can vary little or only for a short time from their normal. Suppose that, by the injection through a needle of a normal saline solution into the vitreous, the intra-ocular pressure be raised, the immediate results will be a diminished inflow by reason of the compressed condition of the uveal vessels, and an increased outflow at the angle of the anterior chamber, till the normal tension has been re-established. Such was Weber's experience, who could not produce glaucoma in this way (24). If, on the other hand, fluid be drawn off from either vitreous or aqueous, the resulting diminution of pressure will produce reduction in the amount leaving the eye, while it will encourage, by permitting dilatation of the arteries, an excessive flow of blood into the interior of the eye with excretion from it at an abnormally rapid rate, till the amount of intra-ocular fluid has reached its average. It is apparent, from what has been said, that the ocular depends materially on the vascular tension.

While all this is apparently simple, the explanation of the origin of increased tension has by no means proved to be so, and our present knowledge of the subject is the result of much labor, observation and discussion. How that has been attained must occupy a very considerable proportion of any study of glaucoma.

It would appear that the increase of the contents of the globe,

relative to its size, the cause of high tension, might result from the retention of too much fluid in the eye, from the exit of too little, or from a combination of these; and the endeavor to determine what is the actual state of matters has resulted in much controversy, which is not yet ended. But, as we have seen, there are several varieties of glaucoma, so different in their apparent origin, that we can not expect to find one explanation which will apply equally well in all its details to each. But, whether or not a common principle underlies the etiology of all, will be seen when we come to discuss "simple," "secondary," and other cases, in contra-distinction to typical, senile, acute or sub-acute glaucoma, which we will consider first.

REFERENCES CONTAINED IN CHAPTER III.

1. Mackenzie—"Diseases of the Eye."
2. Boerhaave—Transl. from "Praelectiones Publicae de Morbus Oculorum"
3. Bowman—Brit. Med. Jour., Oct. 11, 1862.
4. Coccius—"Ophthalmometrie und Spannungs-Messung," Leipsig, 1872.
5. Schweigger—"Ueber Glaucom," Berlin Klin. Wochenschr., 1892, XXIX, p. 657.
6. Fick—Trans. Internat. Congress, Heidelberg, 1888.
7. Koster—Brit. Med. Jour., Oct. 19, 1895.
8. Priestley Smith—Glaucoma, pp. 9, 12.
9. Schoeler—Graefe's Archiv., XXV, 4.
10. Stirling—R. L. O H. Reports, XIII, 1893. p. 419.
11. Deutschmann—Graefe's Archiv., XXVI., 3, p. 117.
12. Schoeler and Uhthoff—Report of Schoeler's Clinic, 1881.
13. Leplat—Annal d'Ocul., Sept., 1887, and Ophth. Rev., 1888, p. 84.
14. Ulrich—Arch. Ophth., XXII, p. 475, and Ophth. Rev., April, 1894.
15. Priestley Smith—Op. Cit. (8), p. 22.
16. Leplat—Annal. D'Ocul., Jan. and Feb., 1889.
17. Schwalbe—Graefe-Saemisch Handbuch, I, p. 50.
18. Gifford—Ophth. Rev., 1886, p. 217.
19. Stilling—Heidelberg Cong., 1885, p. 42.
20. Priestley Smith—"Glaucoma, Its Causes, etc." 1879, p. 142.
21. Schoeler and Uhthoff—Graefe's Archiv , XXV, 4.
22. Knies—Arch. Ophth. N Y., Apr. 1895, XXIV., p. 169.
23. Knies—Virchow's Archiv., LXV., p. 401.
24. Weber—Arch. f. Ophth., XXIII., 1877, p. 1.

CHAPTER IV.

ETIOLOGY (Continued).

HYPERSECRETION VERSUS RETENTION.

Before the circulation of the ocular fluid was well understood, and before the means by which its exit may be interrupted had been made clear, a very natural inference from

the hardness of the eye was that there is in glaucoma excessive secretion, due, as was first thought, to inflammation of the uvea, the inflammatory form being the type of the disease; or, as held by Donders and others after him, the result of abnormal activity of the nerves which govern secretion inside the eye, the "simple" being then the typical form. When Mackenzie (1) noted the relationship between high tension and glaucoma, he concluded that the disease was due to choroiditis which poured out fluid till the resulting pressure destroyed the soft tissues exposed to it, and he was confirmed in this impression by finding in excised glaucomatous eyes that the choroid was disorganized. In 1842 Sichel (2) stated that his dissections never showed any alteration in the vitreous, but that he "had always seen glaucoma succeed a manifest infiltration or at least a congestion, acute or chronic, of the choroid." Arlt (3) observed a choroidal disease in an excised eye affected by secondary glaucoma. Graefe (4), in 1857, held "acute glaucoma to be a choroiditis (or irido-choroiditis) with diffuse infiltration of the vitreous (and aqueous), with a rapid rise of tension through increase in the volume of the vitreous, compression of the retina, and the known series of consecutive symptoms;" and, in 1869, he wrote (5) "The beginning of glaucoma certainly lies in the uvea." . . . "We must admit certain predisposing or coöperating forces, be they in the nerves, vessels or tissues, through which serous choroiditis or irido-choroiditis receives the characteristic stamp of inflammatory glaucoma." Graefe, not being able to reconcile this with the symptoms of simple glaucoma, called the latter "Amaurosis with optic nerve excavation." Brailey (6), at one time looked upon glaucoma as an over secretion from an inflamed iris, ciliary body, and choroid, which resulted rapidly in atrophy of the affected tissues; also, that excess of pigment sometimes in diseased eyes caused high tension. In 1881, having found the *pars ciliaris retinæ* detached from the subjacent pigment layer in ten per cent. of the excised eyes with primary glaucoma examined by him, but never where the tension was normal or decreased, he (7) asked: "does it indicate that fluid is secreted by the pigment layer more rapidly than it can pass onward into the vitreous chamber?" In a later paper, published the same year, Brailey (8) insisted that inflammation of the uvea is always present before the presence of *plus* tension.

Mauthner's Theory

(9) was that glaucoma is a choroiditis, complicated, in the anterior segment of the globe, by inflammatory symptoms, which have their origin in different regions of the choroid, and may thus affect the retina at various points. The result is usually a rapid increase of tension, but the disease may progress without this. It is peculiar, not only on account of the *plus* tension, but also because of "an affection of the optic nerve, which is dependent upon the process in the choroid, but is not always present."

Fuchs (10), who is not a supporter of the secretion theory, reported in 1878 that, in 28 out of 57 glaucomatous eyes, he had seen with the ophthalmoscope atrophic patches in the anterior part of the choroid.

Ulrich

(11) has founded a theory of hypersecretion, with secondary retention due to abnormal impermeability of the iris which also becomes attached to the cornea, and based upon the results of experiments made by him on rabbits, whose cornæ he removed so as to produce complete anterior synechia, as well as upon microscopical examination of pieces of iris removed from human glaucomatous eyes. Histologically, he found the iris atrophic and sclerosed with obliteration of vessels; the ciliary processes hyperæmic, with pigmentary thrombosis, "leading to disturbance of the circulation of the blood in the iris and ciliary body. . . . Immediately following incarceration of the iris in the corneal scar there was chronic hyperæmia of the ciliary processes, and also hypersecretion of the aqueous, the demonstration of which scarcely required the use of fluorescine."

"This hypersecretion is a fundamental factor in the production of glaucoma, and accordingly the entire theory is a *secretive theory*, the same that I advanced in 1884, explaining ordinary inflammatory glaucoma as well as the two principal forms of secondary glaucoma in man. Impeded permeability of the anterior chamber may be an accessory cause." It is a question whether this should not rather come under the heading of retention theories, in spite of the author's distinct assertion to the contrary, especially when one considers that he speaks elsewhere (12) of "the increase in volume of the vitreous pressing the iris and lens forward and together, and cutting off, more or

less, the pupillary route between the anterior and posterior chambers." The iris, he says, "is rubbed against the cornea, becomes hyperæmic and exudes material which binds it to the cornea. This causes the exit by the optic nerve to be more used, which, along with the increased tension, produces cupping. The attack of glaucoma is brought on by all conditions which interfere with the blood circulation, be they general (heart, etc.,) or special to the eye (Mydriasis)." Birnbacher (13). considering Ulrich to have asserted that fluid passes into the anterior chamber in normal circumstances through the base of the iris, and expressing his inability to agree with that statement, Ulrich explained that in glaucoma the pupil is not free on account of the increase in size of the vitreous pushing the lens into closer contact with the iris; and pointed out, in support of the permeability of the iris, that there is fluid in the anterior chamber in total posterior synechia, which could only get there through the iris. To this argument one may reply that Leber said it was an open question whether some of the aqueous was not secreted by the anterior surface of the iris; while we know that the iris is usually bulged forward in such cases; besides which, a little fluid may in certain cases filter through an apparently, but not really, complete posterior synechia, or through an atrophic iris.

The Theory of a Secretion Neurosis

was introduced by Donders (14). He considered inflammatory symptoms to be incidental, and secondary to increased tension. The ciliary branches of the fifth nerve, being irritated, caused a reflex flow of blood with excessive excretion in a manner similar to that which occurs in the glands. The primary irritation was thought to be due especially to senile changes involving the nerves as they passed into the interior of the eye. Magni found the ciliary nerves in glaucoma to be constantly atrophic. Von Hippel and Gruenhagen (15) by irritation of the fifth nerve, and by extirpation of the superior cervical ganglion, produced hypersecretion of the intra-ocular fluid with raised tension, which they held to support the neurotic theory; and Schmidt-Rimpler (16), in 1875, thought that, among other things, irritative conditions in the region of the trigemini were important in influencing the production of glaucoma. Schweigger (17) wrote, "The

fact that nervous irritation, etc., may occasionally provoke glaucoma proves that nervous influences may determine periodical attacks of increased tension."

In considering the former of these theories, evidence has already been adduced showing that a mere increase in the amount of fluid in the interior of the eye produces only a very temporary rise of tension. Glaucoma is not by any means a usual feature of uveal inflammation, and when it does occur it is generally different from common glaucoma, and due to another explicable cause. And glaucoma is common without any sign of such inflammation. It is true that in old glaucomatous eyes, patches of inflammation are frequently met with in the choroid and in the ciliary region, but there is reason to believe these to be among the many secondary changes resulting from long continued excessive pressure, or merely accidently associated with glaucoma. At the same time it is not unlikely that, in uveæ which are already diseased, the congestive or other disordered states, which may start the cycle of events which constitute glaucoma, may be more easily set in motion.

In connection with the theory of a secretion neurosis, little favorable evidence of value has been advanced. Hermann Schmidt (18) expressed a belief that severe toothache produced increased intra-ocular tension; but Priestley Smith (19), showed by the examination of patients in a dental hospital that this view was erroneous. Pain in the fifth nerve, as in any other nerve, might start glaucoma; but probably, neither directly nor reflexly, but rather by inducing exhaustion and sleeplessnes, and its common occurrence in glaucoma can best be explained as a secondary result of the pressure on the nerves within the eye, which is supported by the fact that relief generally immediately appears on reduction of the tension. Weber (20) endeavored unsuccessfully to produce glaucoma by dividing nerves, tying them, passing threads, glass, etc., into the eye, as well as by paralyzing the sympathetic, and irritating the fifth nerve.

Retention Theories.

The discovery of certain pathological changes in the outlets of the now better understood circulatory system for the intra-ocular fluid, along with the apparent insufficiency of the hypersecretion theories, brought into great and

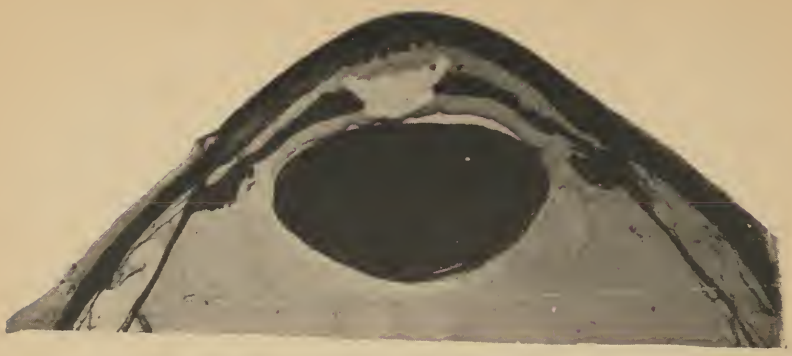

Fig. 3 shows the angle of the anterior chamber, the relationships of the cornea, iris, ciliary body, Schlemm's canal, and lens in a normal eye.

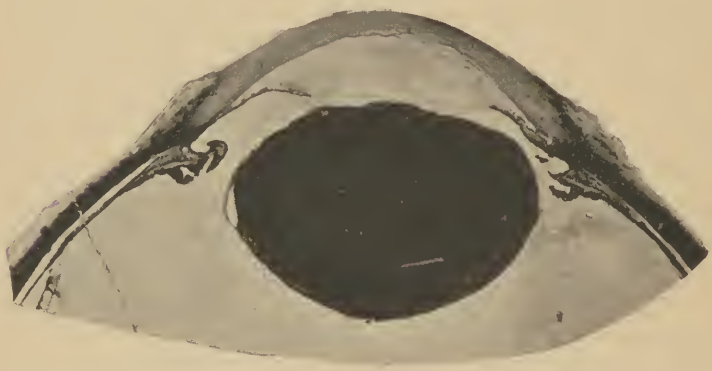

Fig. 4. Primary absolute glaucoma, without operation. The lens was cataractous, and there was cystic degeneration of the retina. This section should be compared with that of the normal eye.

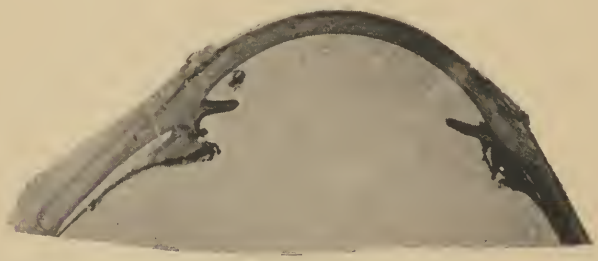

Fig. 5. Glaucoma of long standing, with peripheral anterior synechia, shrinking of iris, ectropion of uveal prigment, and anterior displacement of the sphincter of the iris.

enduring prominence the idea that it is not so much an increased inflow as an obstructed outflow which lies at the bottom of raised tension with all its consequences. Knies and Weber were not the first to notice these changes, but nearly simultaneously they first observed their importance, and brought it before the profession, though not with the same explanation. It has been pointed out by H. Snellen that, though H. Mueller is generally credited with first noticing this condition in 1858, Donders (22) mentioned in 1855 "the diminution of the anterior chamber through adhesion of the periphery of the anterior surface of the iris with the posterior surface of Descemet's membrane, simply as a consequence of increased pressure through exudation processes in the hind part of the eye." Mueller (23) said, in 1858 "The iris adheres with its ciliary border to the periphery of the cornea, whilst it separates very easily from the ciliary body;" and, "the iris adheres with its periphery more closely to the sclera than to the ciliary body, as frequently happens in such cases." Schmidt-Rimpler (24), in 1875, mentioned having observed the peripheral zone of the iris pressed hard against the cornea, but in a summary does not include this among the important points in connection with glaucoma, except in saying that there are possibilities of interference in the outflow of lymph. In 1876, Pagenstecher (25) reported eight eyes, in two of which he had observed that the iris and cornea were in contact. Knies (26) soon afterward wrote an account of fifteen excised glaucomatous eyes, which showed, besides infiltration of various tissues, peripheral anterior synechiæ, and Weber (20), a little later, followed with an account of certain experiments on rabbits, and with four cases in which the same position of the iris was present. Closely behind Weber's paper, came another from Knies (27) reporting six more cases of the same kind, after which the adhesion appears to have become a well recognized condition in glaucoma, and pathologists soon began rather to record cases in which it was absent. The meaning of it has excited much controversy, which has greatly aided the development of a true understanding of the glaucoma process.

REFERENCES CONTAINED IN CHAPTER IV.

1. Mackenzie, "Diseases of the Eye."
2. Sichel, Annal D'Oeul, V., p. 184.

3. Arlt-Zur Pathol. Anat. des Auges "Prager Vierteljahrschrift. 1847, p. 53.
4. v. Graefe Arch. f. Ophth. III., p. 481.
5. v. Graefe Arch. f. Ophth., XV., p. 108.
6. Brailey, R. L. O. H., Rep., 1877, IX., p. 199.
7. Brailey, R. L. O. H., Rep., X., 1881, p. 372.
8. Brailey, R. L. O. H., Rep., X- p. 282.
9. Mauthner, Arch. Opth., N. Y., VII., 1878, p. 178.
10. Fuchs, Heidelberg Cong., 1878, p. 85.
11. Ulrich-Arch. Ophth., N. Y., XXII., 1893, p. 475 and Ophth. Rev. Apr., 1894.
12. Ulrich, Arch f. Augenh., XXII., 1893, p. 1.
13. Birnbacher, "Ein Beitrag zur Anta. des Glaukoma Acutum." Gratz, 1890.
14. Donders-Arch. f. Ophth. IX., 2.
15. v. Hippel and Gruenhagen-Arch. f. Ophth. XIV, 3, XV. XVI.
16. Schmidt-Rimpler, Graefe-Saemisch Handbuch, 1875.
17. Schweigger-Arch. Ophth. N. Y. XX., 1891, p. 475.
18. Hermann Schmidt-Graefe's Archiv., XIV., 1, p. 107.
19. Priestley Smith, Glaucoma, p. 39.
20. Weber-Arch. f. Ophth. XXIII., 1877, p. 1.
21. Snellen, Oph. Rev., Feb., 1891, p. 42.
22. Donders-Arch. f. Ophth. I., 2, p. 75.
23. Mueller-Arch. f. Ophth., IV., p. 377.
24. Schmidt-Rimpler-Op. Cit. (16), p. 61.
25. Pagenstecher-Arch f. Ophth, XXII, 2, p. 271.
26. Knies-Arch f. Ophth. XXII., 3, 1876, p. 163.
27. Knies-Arch. f. Ophth, XXIII., 2, p. 62.

CHAPTER V.

ETIOLOGY—(Continued).

THE THEORIES OF WEBER AND KNIES.

Weber's Theory.

Weber (1A) endeavored to produce glaucoma by artificially exciting a serous choroiditis (Graefe's theory), or through division and irritation of nerves (Donder's theory), but with as little success as he attained by injections into the vitreous. He therefore concluded that the disease had a mechanical causation. He then tried to shut off the exits for the intra-ocular fluid, first by injecting quicksilver into the anterior chamber of rabbits; but this simply destroyed the eyes. He then used oil, which in one case produced a glaucoma-like affection with iritis, anterior adhesion of the iris, and enlargement of the cornea, but without cupping, because the optic nerve of rabbits enters the eye at an angle of 45 degrees. Weber also specially reported four cases in human eyes. These included a secondary and a simple, as well as congestive cases, and, in the con-

gestive and secondary cases, the iris adhered to the cornea, and the ciliary processes were either enlarged or atrophic. He concluded that "in all cases of glaucoma, inflammatory and non-inflammatory, primary and secondary, the filtration passages are narrowed and finally closed," the result of *swelling of the ciliary processes* which come to press upon the iris behind till the latter touches the ligamentum pectinatum and back of the cornea, and cuts off from the angle the fluid in the anterior chamber.

Knies's Theory.

In the cases already referred to which Knies reported in 1876 the points which he specially noted were cellular infiltration of iris, cornea, and choroid, with closure of the angle, and cupping of the optic disc. In 1890 he (1) entered fully into the subject, explaining the connection which appeared to exist between these conditions and the onset of glaucoma. He separated simple glaucoma altogether and said it was another disease. He advanced the theory that in congestive glaucoma there exists an *irritant poison within the ocular vessels alone*, and that this, having passed within the tissues of the eye, sets up an inflammation in the channels at which it has its exit. He defined glaucoma as a distinct and peculiar disease "which begins with cell infiltration of the corneo-scleral border, the neighboring tissue, and with inflammatory symptoms of the optic nerve, going on to adhesion of the periphery of the iris with the cornea, and obliteration of the spaces of Fontana, and if not treated it leads to blinding with characteristic cupping of the optic nerve." He considers the infiltration of tissue round Schlemm's canal to be present before *plus* tension; and, though in many cases the iris is then normal, there are others, especially very acute cases, in which an exudation of white and red blood corpuscles and pigment are found on the anterior surface, sufficient visibly to produce adhesion between iris and cornea. The ciliary bodies he found also infiltrated before the advent of high tension, which is true to a less degree of the ciliary processes. When a case is going to be acute there are very often signs of iritis and cyclitis; when less acute these are wanting, or found only in the angle or ciliary muscle." "One receives the impression that finally the *whole neighborhood of Schlemm's canal* was after this cicatricially drawn in, whereby the iris periphery was made, through

shortening and retraction of the fibres of the ligamentum pectinatum (Brailey) to lie on the cornea and finally to adhere to it. The point of adhesion and cicatrisation shows objectively no longer any sign of cell filtration," though not infrequently some round cells are scattered about. The initial inflammation of the ciliary body soon gives way to atrophy, the circular muscular fibres in this condition turn forward at an acute angle toward the lens (Weber); sclerosis ensues (Brailey); and frequently after the commencement of *plus* tension (or as its cause, according to Brailey, and so before *plus* tension) dilatation of the arteries and thinning of their walls are found. The early congestion with little cellular infiltration existing in the ciliary processes results also, but to a less extent, in atrophy. The processes lie close to the iris, but do not touch it, therefore "Weber finds himself compelled to say that his preparations were much more convincing when fresh." Weber's explanation is not generally applicable, and in typical cyclitis where the ciliary processes are much swollen, the results are not similar to ordinary glaucoma, and the anterior chamber is deep. (See serous iritis.)

When Fontana's spaces are only partially blocked increase of tension may be very slight, but when the blockade is combined with hypersecretion the eye is bound to become harder. The cells about Fontana's space may press the fibres asunder, and on disappearing leave holes through which the fluid may escape, the tension become normal or *minus* while the angle is still blocked. Heightened tension, he said, is not synonymous with glaucoma, but is only a symptom.

When this theory is compared with that of Weber, some noteworthy differences will be observed. Both have proved to be of great service in ocular pathology, not only on account of the main point upon which they agree, the sealing up of the filtration angle, but also because of the stimulus they gave to farther investigation and discussion, which requires to be considered here.

Brailey (2) found in 53 glaucomatous eyes the corneo-iritic adhesion to be generally present, and he admitted the angle of the anterior chamber, uncovered externally by conjunctiva, as normally a likely place for the escape of fluid, and that its closure by impervious iris might without doubt be a factor in keeping up increased tension, while it could not, he thought, be its main cause, for the adhesion

is found without *plus* tension, and *plus* tension is found with-without adhesion. While not denying that apposition between iris and cornea might be primary, Brailey held that it was probably secondary to some mechanical pressure, evidenced by one of his cases where there was near contact and no adhesion, and this, he considered, emanates from behind the plane of the lens, in the form of an increased flow "issuing somewhere between the ciliary muscle and the periphery of the lens," but he did "not think that the advance of the lens will account for it." The adhesion between the iris and cornea, like that between the external and internal surfaces of Schlemm's canal, and like the increase of nuclei in that neighborhood, is merely the result of compression. In his next contribution (3) in 1879 he reported on the pathological anatomy of 149 eyes, some of them considered in the last, but all of them having had their tension accurately recorded at the time of excision. Of these, either the angle of the anterior chamber or Schlemm's canal was closed in some part in all but 8 of the 86 which had *plus* tension, 9 had the angle free above and below; of the 21 which had tension normal 14 had the angle free above and below; of the 34 which had *minus* tension, 18 had the angle free above and below. Schlemm's canal was either quite closed in some part of its circumference, or barely visible, in 111 of the 149 eyes. Signs of inflammation were generally seen where the iris was applied to the cornea chiefly on the anterior surface of the iris and between it and the cornea. In the next paper in 1881 (16) Brailey asserted that the increase of tension follows an inflammation of the ciliary body, iris, and optic nerve, but chiefly of the ciliary body, the enlargement of whose folds "due to their vascular turgescence" is indeed the initial cause of the application of the iris to the cornea, which is nearly always present, and which is continued by pressure arising behind. This may be taken as so far supporting Weber's theory.

In 1877, Wecker (4) denied the inflammatory character of the congestive symptoms of glaucoma, and in 1879 he says (5), "It is now two years since I published in the Archives of Ophthalmology this sentence, 'According to Donders the excess of tension in glaucoma is the result of *hypersecretion*; from my point of view it results in the majority of cases from some check in excretion.' 'The only difference between Weber and myself is that he uses

the word filtration while I use excretion.' . . . . 'In the work quoted above I have alluded to the fact that the channels of filtration discovered by Leber become narrowed by hyaline or epithelial masses,' and 'I was the first to adopt the idea that glaucoma resulted from impaired filtration.' . . . . 'I have always taught that at the optic nerve and angle of the anterior chamber alone the eye is without a hyaloid amorphous membrane, quite unfit for filtration.''

Pagenstecher reported at the Ophthalmological Congress in 1877, six attempts to produce glaucoma in rabbits' eyes by the injection of oil into the anterior chamber, with one success in the production of increased tension, and none in the production of cupping. He also reported cases with *plus* tension and a free angle, and others with *minus* tension and a closed angel.

Schnabel (6) in 1878 examined thirteen eyes enucleated for glaucoma, and found the cornea and iris merely in apposition in one case, adherent in ten cases, and not even in apposition in two which were cases of glaucoma simplex. The longer the *plus* tension, the more firm he found the adhesions, and the iris was always thin at the adhering parts. Schlemm's canal was generally normal and empty, but sometimes narrow or invisible. "I have never seen a connecting substance which could have been supposed to be produced by the agglutinated walls of the chamber or to have preceded or caused the adhesion," and he believes that there is no iritis in common glaucoma. Schnabel considered as antagonistic to the Knies-Weber theory the fact that he had seen eyes lost by uveitis possessing an angle closed by the contact of iris and cornea, "appearing in undoubted inflammation and persisting in soft eyes;" and in eyes recovered after iridectomy where the iris still remained peripherally attached to the cornea, "inflammation does not precede the adhesion at the angle, as Knies says but follows it." "The apposition of the iris and cornea of some duration is the essential cause of the obliteration of the angle of the anterior chamber." . . "The attachment of the iris to the ligamentum pectinatum is caused by a disease of the endothelial lining of these tissues, but I cannot decide whether this disease is only the consequence of abnormal contact or whether, as in kerato-iritis, it is one of the effects of this process." . . . . "It is certain that it is not produced only by the infiltration

of the two surfaces." . . . . "The obliteration of the angle of the anterior chamber alone is harmless; the traction of the origin of the iris, and the stretching of the sclerocorneal margin cause the increase of tension." Schnabel also asserts that the ciliary processes are not necessarily swollen, as shown by a case of his where they had previously atrophied.

Schoeler (7) in 1879 produced *plus* tension and cupping in rabbits by cauterising the corneo-scleral margin. Birnbacker and Czermak (8) considered the adhesion at the angle as a part of the general inflammation of various grades in the uveal tract, and not a mere symptom of pressure; and again Birnbacher (17) described a case which caused him to relinquish the idea of a primary inflammation of the angle.

Schweigger (18) was disinclined to adopt a retention theory, and argued that slight passing attacks controverted it. "In what way does the filtration angle become so quickly blocked up, and so suddenly restored?" If this, as he calls it, Leber's theory, were true, every narrow angle or anterior synechia would produce glaucoma. Reindorf (9) thought that a shallow anterior chamber is unfavorable to the views of Knies, for with the initial blockade at the angle the iris should be pushed back from the cornea.

It will be necessary later to consider a modification of the Knies and Weber theory, first published in 1879, and later built up by prolonged and careful investigations, by Priestley Smith; but in the meantime it will suffice to state that in some 80 glaucomatous eyes examined by himself he always found the angle closed by apposition of iris and cornea, except in three or four cases in which there was special reason for the absence of this feature. To quote, (10) from his book, "The ciliary processes are usually altered, both in size and position. If the glaucoma have been of recent date and congestive type, they are enlarged, their apices extending forward far beyond the customary limit. In such cases they are usually in close contact with the iris anteriorily, and sometimes with the margin of the lens internally; or, if no longer in contact, their wedge-like shape shows that in the living eye they have been tightly pressed between these structures. Viewing them in transverse section we see that they are increased in thickness also, the spaces between the pro-

cesses being narrowed, or even obliterated by the swelling of the lateral convolutions. If, on the other hand, the glaucoma have been of long standing the processes are sometimes much shrunken ard retracted, being then far removed from the base of the iris; but even in such cases the iris base often bears the impress of the former contact. Between extreme hypertrophy, on the one hand, and extreme atrophy, on the other, any degree of swelling or shrinking may be present. The ciliary muscle participates in these changes. At first it is drawn forward with the processes; later it retracts and atrophies." . . . . "Sometimes the iris base adheres to the processes, and is drawn backward when they retract, so that its adhesion with the cornea is more or less torn through; or, if not torn through, the iris base may be stretched in the direction of its thickness, and present a peculiar bend or notch in its anterior surface just at the limit of the adhesion."

Ulrich (12) said that Knies did not explain the origin of the irritating material which he supposes to give rise to the inflammation which originates the corneo-iritic adhesion; and as uveal pigment is evidently carried into the angle, why should not the cells found there have the same distant origin? "It has long been known that in fresh cases of glaucoma the so-called corneo-iritic adhesion is only a simple apposition, and not a true adhesion." Anatomical conditions and laws of physics render it untenable that the anterior chamber should be shallow were the angular adhesion primary, but it is quite otherwise when the retention is behind the iris.

Galezowski (13) in 1894 had "almost always, if not always, found changes of the canal of Schlemm, of the lymph channels in the sclera, of the choroid, and of the optic nerve itself, whence arises the conviction that in the obliteration, partial or total, of the canal of Schlemm, resides the principal cause of glaucomatous affections." He quoted two cases of glaucoma, secondary to dislocation of the lens, which seemed to show that the high tension was produced by the lens pressing forward against the iris, the tension diminishing on the head being thrown back.

Bitzos (14) wrote, "It is necessary to reject all theories explaining glaucoma by localizing the lesion in the sclera, choroid or zonule, for several reasons; above all because they do not explain the propulsion of the iris. One is thus

compelled to find the cause behind the lento-zonular diaphragm."

Knies (15) has recently recorded a series of experiments on the eyes of dogs with the avowed object of demonstrating "the passages of outflow from the vitreous and aqueous by means of the changes which substances capable of provoking irritation and inflammation excite in their passage through the walls of the eye-ball." It is apparent from this paper that its author still holds that the adhesions at the angle of the anterior chamber are due to irritation set up by what might be termed a specific glaucoma poison, as it passes out of the eye, and he endeavors to reconcile all the results of his experiments with this idea; but, as the unprejudiced observer must admit, with indifferent success. He first, as he did before, overcomes the difficulties which confront him in connection with simple glaucoma by putting it entirely out of court, on the very doubtful ground that it is not glaucoma at all; and then, confining himself to inflammatory cases, calls the disease "Irido-cyclitis in which the anterior channels of outflow of the eye are occluded, temporarily at first, afterward permanently." This is to be distinguished from ordinary Irido-cyclitis "by the fact that in the latter the nocuous element has from the outset its seat in the iris and ciliary processes, while in the former it circulates in the vitreous and aqueous, and in its exit from the eye causes an inflammation of the iris and ciliary body starting from the region of the angle of the anterior chamber. It must be admitted that in the genuine glaucomas we can make no definite statements in regard to the nature of this element; while in the secondary glaucomas, *i. e.* in those for which we know an exciting cause, whether it be a malignant tumor, a luxated lens or retinal haemorrhages, we must assume an existence of such an element circulating in the vitreous." But no such hypothesis is really required, for glaucoma in all these and other conditions, has been explained in a manner which is simpler, more evident, more inclusive, and which does not necessitate the designation of the quieter forms by another name.

Knies introduced into the vitreous (a) olive oil, (b) dilute oil of turpentine, (c) strong oil of turpentine, and (d) a mixture of unguentum hydrargyri cinereum, olive oil, and oil of turpentine. In every case in which dilute oil of turpentine was used an attack of glaucoma set in about

six hours later, and Knies claims this as the first series of experiments in which glaucoma has been artificially produced. (See Weber, p.38, and Pagenstecher, p. 37.) The attacks, which were all transitory, were characterized by the usual signs: marked increase of tension, mydriasis, and sometimes slight vapor-like opacity of the center of the cornea." That the glaucoma was transient is explained on the hypothesis that an albuminous excretion, apparent, though slight, coagulation in the vitreous and aqueous blocked up the channels of excretion, and afterward contracted so as to permit of the passage of the aqueous. Knies does not say that the anterior chamber was shallow, nor does he explain how a mere blockade of the channel of exit, even with a subsequent local inflammation, could produce such a condition, which on the face of it appears to be impossible; while, if the anterior chamber was deep, the *plus* tension might also have been due to the filling of the angle with the injected oil.

The strong oil of turpentine proved too irritating and was abandoned, and the simple olive oil turned out to be infectious and had disastrous results of no value in this connection. The "oleum cinereum" acted in a much more active manner than the dilute oil of turpentine, and produced inflammatory reaction, what Knies called "glaucoma imminens" without increased tension (in fact not glaucoma at all), "since the action of the liquids used by me was obviously different in a qualitative way from that of the products of tissue metamorphosis that are the efficient agents in human glaucoma." . . . "Obviously, in order to insure the development of a fully fledged glaucoma, the inflammatory agent *must act continuously, but less intensely* than the material that I used, *i. e.* must probably act like a substance secreted by a growing malignant tumor."

REFERENCES CONTAINED IN CHAPTER V.

1. Knies, Centrabl. f. Allgem. Path. I., p. 249.
1A. Weber, Arch. f. Ophth., XXIII., 1877, p. 1.
2. Brailey, R. L. O. H. Rep. IX., 1877, p. 199.
3. Brailey, Op. cit. (2) p. 380.
4. Wecker, Arch. f. Ophth., XXII., 4, p. 209.
5. Wecker, "Ocular Therapeutics," London Ed., pp. 250, 260.
6. Schnabel, Arch. Opth., N. Y., VII., 1878, p. 12.
7. Schoeler, Arch. Ophth., XXV., p. 63.
8. Birnbacher and Czermak, v. Graefe's Archives, XXXII, 4.
9. Rheindorf, Klin. Monatsbl. f. Augenh. XXV.
10. Priestley Smith, "Glaucoma," p. 78.
12. Ulrich, Arch. f. Augenh., XXVI., pp. 1-19.
13. Galezowski & Chauvel, Bull. de l'Acad. de Paris, XXXI., p. 240.
14. Bitzos, Annal d'Ocul, CXII., pp. 92-103.
15. Knies, Arch. Ophth., N. Y., XXIV., 2, 1895, p. 169.
16. Brailey, R. L. O. H. Rep., X., 1881, p. 282.
17. Birnbacher, "Ein Beitrag zur Anat. des Glaukoma acutum," '90.
18. Schweigger, Arch. Ophth., N. Y., XX., p. 475.

CHAPTER VI.

Etiology—(Continued).

THE THEORIES OF BRAILEY, OF PRIESTLEY SMITH AND OF RHEINDORF.

Brailey's Theory.

Between the years 1877 and 1881 W. A. Brailey, then curator of the laboratory of the Royal London (Moorfields) Ophthalmic Hospital, published a number of papers on the subject of glaucoma, and reported the examinations of a very large number of eyes generally excised for pain, ulceration, or some other complication incident to the later stages of the disease. It is interesting to follow the development of the opinions on its etiology of one so favorably situated for personal observation, and in touch with the literature of the times.

In 1877 he (1) began by examining 53 cases of which, however, 33 were secondary and can be dismissed for the present. In the remaining 20 he noted chiefly the condition of the ciliary body and the choroid, and the former he found exhibiting pathological signs in 14 cases, while both ciliary body and choroid were affected in 5, and in only 1 the choroid alone, the ciliary body being healthy. Besides, he observed in every case except 1, that the ciliary muscle, and especially its circular fibres, were atrophied, and that to a degree corresponding to the amount of increased tension, rather than to its duration. Concerning this observation Schnabel (2) wrote in 1878, "It is Brailey's great merit to have first observed and described the atrophy of the ciliary muscle. This is the only anomaly in the glaucomatous eye demonstrated hitherto by anatomical examination, which has undoubtedly a relation to the glaucoma process, and is not merely a consequence of it. It exists before the glaucoma can be clinically diagnosed, for I found it very well developed in a globe in which the increase of tension had existed for only a few hours." At this time Brailey, evidently taking the inflammatory signs in both the ciliary body and choroid for primary changes, expressed his belief that the high tension is the result of hypersecretion from one of these, and in view of the condition of the ciliary muscle suggested the presence of "some form of inflammation resulting rapidly in atrophy, whether affecting the iris, ciliary body, or choroid," while he looked upon the peri-

pheral irido-corneal adhesion, which was commonly present, as of only secondary importance. In his continuation of the same subject in 1879, he reported the examination of 149 eyes, which had all been tested and noted as to intra-ocular tension at the time of the excision. Some of these were included in his last paper. In this he paid special attention to the ciliary muscle, and took up the subject of the *circulus arteriosus iridis major*, which appeared to him abnormal in glaucoma. He found on making careful measurements that in eyes with increased tension the average diameter of the artery was not quite twice the normal, and that its average wall thickness was about half the normal, while the ciliary muscle was only about two-thirds the normal size, except in cases of "shortest standing" in which, unlike Schnabel, he says this is not found. On the other hand, where diminished tension existed in glaucomatous eyes the condition of both was normal, which must strike one as peculiar, seeing that such eyes had probably previously suffered from increase of tension. Brailey does not connect the condition of the artery with backward pressure, because the veins are not similarly affected, neither does he look upon the ciliary atrophy as due to pressure, he having seen two cases in which it is quite localized, but he puts it down as second, in point of time, to the arterial changes. In these eyes, too, the angular adhesion is common. Knies said of the dilatation and thinning of this artery before the tension is increased, as described by Brailey, that it is not constant, and that it is seen, though less often, in both normal and diminished tension; while v. Garnier (4) found diminution, instead of increase, in its diameter. The atrophy in the ciliary region at a late stage has been generally recognized. For instance, Birnbacher and Czermak (5) found it in all the seven cases introduced in their paper. Knies (6) considered atrophy and sclerosis of both ciliary muscles and processes to be common as a rapidly developed sequel to an infiltration preceding increased tension, and Treacher Collins (7) described it as a usual thing in the eyes excised for prolonged glaucoma, which are to be seen in the museum in the Royal London Ophthalmic Hospital. By the time of his next contribution in 1880 Brailey (8) had advanced the hindrance to the outflow of lymph to a position more closely approaching in importance that of increased inflow. The latter of these two elements which

contribute to the causation of glaucoma is due to inflammation, or visibly increased blood supply; while the former is generally the result of the blockade at the angle, and rarely of an obstacle at the exits from the perichoroidal space; while he found "in most cases such a condition of the optic nerve as would hinder the passage of any fluids along it." He attached special importance to the early atrophy of the ciliary body, because he believed that that permits the more easy approximation of the iris and cornea, whereby the former acquires a valvular action in relation to Schlemm's canal, which is potent "under the influence of the normal or even slightly diminished flow" from the angle of the anterior chamber. Knies (6) found that the atrophied ciliary body can be pushed abnormally easily back and forward.

The above may be considered as Brailey's explanation of "anterior," while the following is that of "posterior" glaucoma, a division first proposed by the younger Desmarres, and indorsed ten years later by Wecker, Stilling, and some other authorities: In less common cases an increased secretion arises from a pathological condition "showing accumulation in the intergranule layer near the optic disc, or more often in the neighborhood of the ora serrata; besides accumulation in front of the lamina cribrosa * * * and sometimes * * * a fissure or layer of loose tissue leading to this from the intergranule layer. * * * This is consistent with a current from the outer retinal layers of the lymph channels of the optic nerve." When to this increased inflow there is superadded a hindered outflow, due to sclerosis of the optic nerve and lamina cribrosa, there is sufficient reason for an increase of intra-ocular tension. (This may be compared with Mauthner's theory.)

Brailey's next paper in 1880, which was written in conjunction with Edmunds (10), corroborates the contents of the previous one concerning the condidion of the ciliary arteries and the ciliary body, and goes so far as to assert that in all cases of primary and secondary glaucoma, with increased tension, they found sclerosis of the optic nerve and its vessels, including the central artery of the retina. Brailey's sclerosis of the nerve vessels Knies (6) believes to arise at a late stage, and to be inconstant. But Brailey and Edmunds found the nerve still inflamed and swollen, the sheaths participating, and they express their

belief that in primary glaucoma optic neuritis precedes increased tension. In the locality of the angular adhesion the iris was generally atrophic, but in fresh cases they had often seen it inflamed. The manner of the production of the adhesion they further elaborated by stating that a contraction of the fibres of the ligamentum pectinatum brings the cornea and iris into apposition, which opinion is confirmed by Knies (6). In Brailey's next paper (11) he reported on 92 cases of high tension. In these the angle was generally closed, the ciliary muscle usually inflamed, atrophic, or sclerosed; the *circulus arteriosus major* very frequently dilated, and the optic nerve almost always either inflamed or sclerosed. A description follows by Webster Fox and Brailey (12) of a "case of glaucoma preceded by optic neuritis." They found the discs swollen, and in their opinion "a neuritis caused an acute inflammation of the walls of the central vessels with a resulting thrombosis of the vein;" and this inflammation spread forward to the ciliary muscle. In his last paper (13) Brailey again spoke of the angle being closed on account of forward pressure of the iris from "enlargement of the ciliary folds due to vascular turgescence," Weber's idea; whereas he had recently asserted it to arise from contraction of the fibres of the ligamentum pectinatum, in which Knies had borne him out. Whether, in the meantime, he had changed his mind or thought both correct according to stage and circumstance, I am unable to say, but he again asserted that before the advent of increased tension inflammation of the ciliary body, iris, optic nerve, and, above all, in and around the fibres of the ciliary muscle, is always present, and that along with enlargement of the ciliary folds dilatation of the circular artery takes place, which does not decrease with the atrophy and recession from the iris of the ciliary body. The changes in the choroid are not generally primary infiltration but atrophy from compression. Brailey was now less certain that the dilatation of the circular artery might not, at times, be due to obstructed circulation the result of high intraocular tension, and while he still considered the first step toward this tension to be hypersecretion, he admitted that when atrophy of the ciliary body and iris are present the angular obstruction alone is to blame.

Priestley Smith's Theory.

In Priestley Smith's opinion the causes of glaucoma in general are "extremely numerous, and include such widely different agents as rheumatism, gout, syphilis, anæmia, and many other constitutional diseases; injuries of various kinds; cardiac, vascular, and nervous disorders; neoplasms, congenital malformations, and senile changes. * * * Primary glaucoma seems to depend sometimes chiefly on systemic, vascular, or nerve disturbances, sometimes chiefly on individual peculiarities in the build of the eye, and often on a complex co-operation of several causes"(14). At the same time, by means of laborious investigations, he has been able to sustain a theory publicly broached by himself in 1879 (15), which gives to the relative development of lens and globe a most important place among the conditions which predispose to the disease and make the above named disorders potent to produce it, while in a more healthily constructed eye they might have caused nothing worse than perhaps a passing ciliary congestion. Priestley Smith began in 1880 by measuring the lenses of five pairs of healthy eyes, and later "156 lenses removed from dead subjects were examined. They belong in nearly equal numbers to the six decades of life between twenty and eighty, and in smaller number to the decade eighty to ninety. Each lens was accurately weighed, and then measured as to its volume, by means of an apparatus devised for the purpose. In most cases the linear dimensions were measured also. Opacity when present was noted. The specific gravity was calculated in each case from the weight volume." The following are the results arrived at: The "crystalline lens, so long as it remains healthy, increases in weight and volume throughout the whole of life. During the forty years between twenty-five and sixty-five years of age it adds about one-third to its weight and one-tenth to its diameter. The specific gravity appears to vary a little in individual cases, but shows no decided change with the advance of life. Lenses which are becoming cataractous are, as a rule, smaller than healthy lenses belonging to the same period of life. These are anatomical facts; physiology explains them. The lens is derived from the cuticular epiblast, and in its mode of growth is analagous to the cuticle. But its cells, unlike those of the cuticle, are not

cast off as they grow old; they are laid down layer upon layer within a closed capsule, the younger fibres surrounding the older. In consequence of this unique arrangement, and in spite of the shrinking of the older cells which form the nucleus, the growth of the lens does not cease with that of the rest of the body, but is continuous unless some morbid process intervene, throughout the whole period of life. In advanced life, the process of the growth often fails. Then the shrinking nucelus tends to separate from the softer cortex, and senile cataract begins. Accordingly the lens with incipient cataract is usually smaller than a healthy lens of the same age. To this rule, however, there are perhaps some exceptions; in certain cases of the development of the cataract the softened cortex appears to swell" (16). The obvious effect of the peculiarly sustained growth of the lens, while the tissues which environ it cease to grow at the commencement of adult life or earlier, is a progressive diminution of the intervals between lens and cornea and between lens and ciliary body. If this have anything to do with glaucoma, glaucoma ought to become commoner with advancing years. After careful examination into the matter in 1000 cases of glaucoma, Priestley Smith found that not one per cent. begins earlier than the twentieth year, that its frequency increases, slowly at first, more rapidly later on, in each decade until the sixtieth year; that between sixty and seventy it is about as frequent as between fifty and sixty; that after seventy it frequently diminishes; that cases beginning after fifty are about twice as numerous as those beginning before fifty, and that the liability is more than twice as great between sixty and seventy as between forty and fifty. The reasons given by Priestley Smith for the diminished number between seventy and eighty are the unwillingness of such old people to travel, that many are in workhouses, or ill; to which might be added the probability that any patient liable to glaucoma would be attacked before that age, and be registered under one of the other decades. He also found that, on the whole, females suffer in rather larger number than males, as six is to five, though the chronic non-congestive form affects the latter rather more than the former. Priestley Smith, having shown that the liability to glaucoma increases *pari passu* with the growth of the lens, proceeded to prove that it is still farther increased when this

is associated with an abnormally small globe. He found that, on the average, the size of the cornea is a fairly good index to the size of the globe. The size of the cornea he measured in 250 healthy males and in 250 females, or 1000 eyes, at all periods of life from five to ninety, with the following results: Its average horizontal diameter is 11.6 m.m., and this increases little, if any, after five years of age, but may, perhaps, diminish slightly after forty years of age; it is slightly greater in males than in females; it is the same in hypermetropic, emmetropic and myopic eyes, and even in high ametropia; "it may safely be asserted that the size of the cornea bears no definite relation to the refraction of the eye" (17); there is very rarely any difference between the two corneæ of the same individual, even when the refractions are quite different.

In primary glaucoma, in 216 eyes, the horizontal diameter of the cornea is, on the average, half a m.m. smaller than that of an average eye, therefore the globe is smaller in glaucomatous than in healthy eyes. The percentage of unusually small corneas—measuring 10 m.m. or less—is much greater in the glaucoma than in the healthy group, *e. g.*, a horizontal diameter of 10 m.m. was found nine times in the glaucoma group, *i. e.*, 4.17 per cent. of the eyes, and was not found once among the thousand eyes of healthy persons. In 1894 he asserts (18) that eyes in which the cornea is only 10 m,m. in diameter seldom escape glaucoma.

Priestley Smith also proved that the small size of the cornea is not a result of glaucoma, but preceded it.

Confirmation of the conclusions drawn from examination of the corneæ, that small eyes are more liable to suffer from primary glaucoma than large ones, is given by his *direct* measurement of the globes of 14 eyes blinded by primary glaucoma, in which he found the average diameter in every direction to be fully 1 m.m. below the normal average. But if it were found that these small eyes contained relatively large lenses his argument would be strengthened. To arrive at a correct conclusion on this point he examined his tables of measurements, and found "that the size of the lens bears no fixed proportion to the size of the globe, even at the same period of life, and that in the glaucoma group there are several instances of a large lens in a very small eye; but the measurements are too

few to justify any general statement." As the best evidence he could produce, Priestley Smith gave a series of photo-zincographs of six healthy eyes arranged beside six eyes blinded by glaucoma. In the latter the following features are visible in some or in all: A small globe; a disproportionately large lens; a consequent want of space between the lens and its surroundings; a shallow anterior chamber; a closed filtration angle; an atrophied ciliary body; an excavated optic nerve. To this evidence might, perhaps, be added that of a drawing by Treacher Collins (19) of a microphthalmic eye in which the dislocated lens appears large in proportion to the size of the globe. Priestley Smith, in confirmation of his belief that microphthalmic eyes with transparent lenses are very prone to glaucoma, quotes a case examined by Hocquard and Masson (20) in which the lens was found to be much too large for the eye; or, "speaking more correctly, the eye was much too small for the lens;" a case published by Lang, concerning which Treacher Collins (21) noted that "the interior of the eyeball was filled with lens, which, in proportion to the rest of the eye, was large and more circular than usual," and observed that the same thing is to be seen in the diagrams of microphthalmic eyes published by Kundrat (22). "Evidently an imperfect development of the tunics does not necessarily affect the lens in equal degree, and this, indeed, is what we might expect from the different origin and independent growth of the latter" (23). We are apt, then, according to Priestley Smith, to have in eyes which become glaucomatous, and from the causes enumerated, an abnormally limited space, or none, between the lens and ciliary body, while the iris approximates to the cornea in an unusual degree, a condition of things easily susceptible, by a mere accentuation, of setting up the train of symptoms which together constitute glaucoma. Thus, "any disturbance which congests the ciliary processes, or displaces the lens forward, or thickens the iris, is apt in a predisposed eye to cause a dangerous compression of the filtration angle" (24); and, clinically, we find that "the common antecedents of glaucomatous attacks are exposure to cold, constipation, hunger, sleepliness, bodily and mental fatigue, heart weakness, bronchitis, hepatic congestion," (injury to the eye or its neighborhood) "in short, various conditions which disturb the circulation and tend to congest the

venous system. * * * Moreover, the influences under which the milder attacks subside spontaneously are pre-eminently those which tend to relieve venous congestion, namely, warmth, rest in bed, sleep, purgation, food, etc." That acute glaucoma is not inflammatory in its origin was stated some thirty years ago by Sir William Bowman, more recently by Wecker and others, while the late George Critchett likened it to a strangulated hernia, which resemblance was noticed separately by Priestley Smith, and is also mentioned by Fuchs. "The suddenness with which the earlier and milder attacks appear and disappear, the fact that a drop of atropine solution can excite a violent attack in an eye which has not previously shown the slightest sign of inflammation, and that a surgical operation can at once and permanently put an end to the process, are strongly opposed to such a doctrine" (25).

The theory known as Priestley Smith's is then, in brief, that a congestion of the uvea and especially of the ciliary processes produced by any of the conditions already mentioned, and causing these to swell so as to diminish the capacity of the globe, drives away the aqueous more rapidly at the angle of the anterior chamber, and so permits of the advancement of the lens and ciliary processes which press the iris forward; the swelling also by trespassing on the limits of the perilenticular space causes an obstruction to the outward flow of lymph, which raises the pressure in the vitreous; this still further blocks the exits by more firmly sealing up the angle; a certain permanency of this situation produces, simply as the effect of prolonged contact, an adhesion between the iris and the cornea, upon which, as a result of high intra-ocular tension, the various pathological changes of the optic nerve, choroid, etc., ensue.

But while the above is a sketch of what is generally known as Priestley Smith's theory, it should be observed that he does not consider it to apply, cut and dried, to every case of glaucoma. For instance, he says (26): "Changes in the hyaloid membrane, in the stroma of the vitreous, or in the constitution of the vitreous fluid, are possible causes of diminished filtration, and, as a fact, in eyes blinded by glaucoma the vitreous is often more distinctly membranous than in healthy eyes, its septa being thickened, or coated by albuminous coagula. In some instances, however, the excess of fluid appears to

collect not in the vitreous but immediately in front of it, causing a wide separation of the hyaloid membrane from the ciliary body. When the ciliary processes are squeezed between the lens and the iris, any secretion which they still emit must pass backward, and in this way the blockade must tend to aggravate itself." As will be seen when discussing glaucoma in aphakia, in aniridia, after cataract extraction, etc., many cases, which have been arrayed against the theory, have been found upon close investigation rather to confirm than to annul its general tenets.

Now, let us see what others have written on this subject. As early as 1887 Brailey (1) wrote that the cause of the closed angle is an increased flow from behind the plane of the lens, issuing somewhere between the ciliary muscle and the periphery of the lens, and that the cause must be a mechanical one, as he had seen the iris applied to the cornea without adhering to it; but he did "not think that the advance of the lens will account for it." In 1881, without mentioning Priestley Smith, but evidently with reference to his theory, Brailey (13) remarked that in no one of the specimens which he had examined did the lens exceed the normal diameter, and that in most it was considerably flattened antero-posteriorly. He had rarely seen it advanced, and the posterior chamber of the aqueous was usually of great depth. Nothing, however, was stated concerning what exact methods he employed in estimating the relative size of the lens, and it is to be noted that Priestley Smith never claims a *positive* increase in its size, and, also, that he specially states that the lens may, after excision of the eye, return to a normal position from diminuition of the pressure behind it. In the same year, and with the same lack of information concerning his methods of procedure, he declared (28) that the diameter of the lens, whether clear or cataractous, "is even somewhat below the normal at the corresponding period of life;" that after excision the position of the lens is generally normal, but sometimes advanced; and that the only cases where it is even probable that *plus* tention is due to mechanical obstruction by an enlarged lens are those in which the latter is swollen from traumatism. He stated, however, that the channels between the ciliary folds equal 75 m.m. in width at their widest part, and normally touch the lens. Fuchs (29)

gives the distance between the lens and ciliary processes as .5 mm. The folds are, according to Brailey, larger in low tension than in high. He sums up by saying: "I fail to recognize in cases of primary glaucoma any obstruction to the normal outflow of fluid at the anterior surface of the vitreous sufficient to advance the lens." Brailey (30) in 1890 published a case of a girl, 18 years old, with, in the right eye, M. 12 D. and a cornea of 9 m.m. diameter, the iris absent except a narrow crescentic piece on the inner side for two-fifths of the circle, the lens *in situ* and of a diameter equal to that of the cornea, the optic disc cupped, myopic crescent, opaque nerve fibres, and T. + 1.

Natanson (31) said that, in normal place and continuity, the lens plays a very subordinate role in the pathology of glaucoma, because glaucoma may be primary in aphakial eyes, as in two with which he had met where both eyes were aphakial and both glaukomatous, and another case in which one eye was aphakial and both glaucomatous (see section on Aphakia).

Knies, in 1890, while asserting that in glaucomatous eyes the lens is not pressed forward, quoted Weber to the effect that its propulsion toward the cicatrix after an iridectomy can frustrate the benefits of the operation, and Pagenstecher to the effect that Petit's canal is sometimes widened.

Schweigger (32), in 1891, said that he did not believe that large ciliary bodies have anything to do with glaucoma, and that the crowding forward of the lens, iris, etc., against the cornea are the result of the increase of the vitreous, and not the cause of the disease. He thought also that glaucoma in aphakia witnesses against the retention theory.

Ulrich (33), in 1892, writes that before Priestley Smith's measurements of the lens can be accepted they must be confirmed by others.

Wagenmann (34), in 1892, found propulsion of the lens in each of his three cases of hæmorrhagic glaucoma.

Bitzos (35) rejected all theories which do not place the difficulty behind the lento-zonular diaphragm, because neither age nor aphakia prevents glaucoma.

H. Snellen (36) considered that in advanced life, the elasticity of the lens being gone, and the chief effect of cont. action of the ciliary muscle being then merely to slacken the zonule, "the ciliary processes are thereby

pressed against the lens," and as Priestley Smith says, "the lens may then move forward carrying the iris toward the cornea." Snellen believed that the zonule is sometimes abnormally lax on account of degenerative changes often associated with senile cataract, which might account for some cases of glaucoma associated with an opaque lens, even though the latter were below the normal size, as is apt to be the case. An example of such is given by Priestley Smith (37), where the lens lay almost in contact with the cornea, and probably during life brought the iris into touch with it. (See also section on Accommodation and Glaucoma).

In 1894 Panas (38) wrote, without farther particulars, that, as a result of his dissections and those of Knies, "anatomical pathology has shown us the persistence of a sufficient space between the border of the lens and the ciliary processes, and the absence of all propulsion of the lens."

Treacher Collins (7) in reviewing the anatomical conditions found in the glaucomatous eyes belonging to the museum at Moorfield's Hospital, stated that in many of the specimens the lens is rather larger than normal, or is relatively larger than other parts of the eye. In two cases the ciliary processes are in contact with the sides of the lens, but in most they are atrophied (the eyes having usually suffered for a long period before excision), though at one time they may have touched the lens. In 1894 he (39) said, "of all the theories which have been put forward to explain the increase of tension in glaucoma, that which attributes it to the obstruction of the exit of the aqueous humor from the eye, and which has been so ably expounded and supported by Mr. Priestley Smith, is the most fascinating and explains best the phenomena met with.

Bader (40), when curator in the Royal London Ophthalmic Hospital, in reporting on iridectomy operations during 1857–1859, said "glaucomatous eyes are generally small."

Brugsch-Bey reported in 1886 some independent observations made in Cairo by himself on the eyes of Egyptians. "He found," in Priestley Smith's words, "the average diameter of the cornea to be smaller than in Europeans; he found it to be smaller in eyes suffering from primary glaucoma than in healthy eyes;

and he found that primary glaucoma formed a larger per centage of ophthalmic cases than is usual in Europe." He also quotes Professor Rampoldi (42) as having observed glaucoma more frequently in small eyes than in large ones.

In connection with Priestley Smith's, we may glance at a **theory advanced by Rheindorf** (43) in 1891, and founded on an observation by Samelsohn (44), who watched the color from an iron splinter in an otherwise healthy eye pass through Petit's canal to the equator of the lens, thence through the lens to the anterior pole, back to the insertion of the zonule into the lens, and out into the posterior chamber. Rheindorf thought that the lenticular sclerosis incidental to age, especially when accompanied by something similar in the zonule, acts as a sufficient obstruction to the forward flow to produce glaucoma simplex; while, when superadded to this is any local inflammation or change in the blood current, the traction which he supposes to result to the ciliary body on account of the above senile changes may be sufficient to set up the acute form of glaucoma. He asserted that if the perilenticular space were merely narrowed by enlargement of the lens or ciliary body the only effect would be to increase the rapidity of the passage of fluid in the same way that on the removal of one kidney filtration proceeds more rapidly through the other. In connection with Rheindorf's theory, it will be sufficient to remark that Samelsohn's observation is not in accordance with what are believed to be the most satisfactory explanations of the lymph paths through the interior of the eye; that it would be difficult to reconcile it with certain forms of glaucoma, *e. g.*, glaucoma before the advent of senile degenerations; and that the author of it admits that he well knows that it is hypothesis alone. He believes secondary to be quite different from primary glaucoma, and to be due to irritation of the uvea producg hypersecretion.

REFERENCES CONTAINED IN CHAPTER VI.

1. Brailey—R. L. O. H. Rep., IX, p. 199.
2. Schnabel—Arch. Ophth. N. Y., VII, 1878, p. 12.
3. Brailey—Op. Cit. (1), p. 379.
4. Garnier—Arch. f. Augenh. XXV, 1892, p. 24.
5. Birnbacher and Czermak—Arch. f. Ophth. XXXII, 2, p. 1 and 4, p. 1.
6. Knies—Centralbl. f. All. Path. 1890, I, p. 249.
7. Treacher Collins—R. L. O. H. Rep. XIII, p. 482.
8. Brailey—R. L. O. H. Rep. X, p. 10.
9. Wecker—"Ocular Therapeutics," 1870.
10. Brailey and Edmunds—R. L. O. H. Rep. X, p. 86.
11. Brailey—R. L. O. H. Rep. X, p. 90.
12. Webster Fox and Brailey—R. L. O. H. Rep. X, 2, p. 205.
13. Brailey—R. L. O. H. Rep. X, p. 282.
14. Priestley Smith—Ophth. Review, XIII, p. 225.
15. Priestley Smith—"Glaucoma," etc., 1879, p. 158.
16. Priestley Smith—"Glaucoma," 1891, p. 84.
17. Ditto, p. 99.
18. Priestley Smith—Ophth. Review, XIII, p. 220.
19. Treacher Collins—Trans. Brit. Ophth. Soc. XIII, 1893, p. 117.
20. Hocquard and Masson—Arch. d'Ophth. 1883, p. 231.
21. Treacher Collins—R. L. O. H. Rep. XII, p. 292.
22. Kundrat—"Ueber die angeborenen Cysten im unteren Augenliden, Microphthalmie, und Anophthalmie," Vienna.
23. Priestley Smith—Op. Cit. (16), p. 108.
24. Ditto, p. 124.
25. Ditto, p. 125.
26. Ditto, p. 130.
27. Brailey—R. L. O. H. Rep. X, p. 130.
28. Brailey—Op. Cit. (27), p. 372.
29. Fuchs—"Lehrbuch der Augenh." p. 408.
30. Brailey—Trans. Brit. Ophth. Soc. p. 139.
31. Natanson—Inaugural Dissertation, Dorpat, 1889, and Ophth. Rev. IX, p. 140.
32. Schweigger—Arch. Ophth. N. Y. XX, p. 475.
33. Ulrich—Arch. f. Augenh. XXVI, p. 1.
34. Wagenmann—Arch. f. Ophth. XXVIII, pp. 213, 260.
35. Bitzos—Annal. d'Ocul. CXII, p. 92.
36. Snellen—7th International Cong. Heidelberg, 1888, and Ophth. Rev. Feb. 1891, p. 47.
37. Priestley Smith—Op. Cit. (16), p. 131.
38. Panas—"Maladies des Yeux," Vol. I, p. 516.
39. Treacher Collins—Lancet, Dec., 1894.

40. Bader—R. L. O. H. Rep. II. p. 229.
41. Brugsch Bey.—Ophth. Rev. V, p. 319.
42. Rimpoldi—Trans. Inter. Med. Cong. Washington, 1887, III. p. 752.
43. Rheindorf—Klin. Monatsbl. f. Augenh. XXV and XXIX, p. 33.
44. Samelsohn—Klin. Monatsbl. f. Augenh, 1881.

CHAPTER VII.

ETIOLOGY—(Continued).

Diseased Vortex Veins as a Cause of Glaucoma.

According to some eminent authorities interference with the circulation through the vortex veins may produce glaucoma, and experiments and pathological research have been cited in support of this view. The following are the results which have been obtained experimentally by blocking the lumen of these veins.

Exner and Leber (1), after their ligature, found a quick and decided increase of intra-ocular tension; Weber (2), a temporary, though distinct increase, with congestion of intra-ocular vessels; Schnabel (3), that the fluid oozes through the channels in the sclera in which the vortex veins run, after injections are made into the vitreous in enucleated eyes; Priestley Smith (4) asserted that "ligature of the vortex veins outside the eyes may raise the pressure to twice its normal height," while he said (5) "the lymph passages which, according to Schwalbe, surround the vortex veins and convey lymph from the inner to the outer surface of the sclera, appear to have no connection either with the vitreous or the aqueous chamber. The fluid which is secreted by the ciliary processes after subcutaneous injection in living animals, and which can be traced through the chambers of the eye, by its artificial coloration, has not been found in these channels. The fluid which sometimes escapes through the vortex veins during injection of the anterior chamber in the excised eye appears to reach them through the veins of the iris; it never passes between choroid and sclera."

Schoeler (6) found that after occlusion of all the vortex veins in rabbits' eyes, with a hot iron, the appearances already found by Leber, Weber, and others, were produced with chemosis, sometimes exophthalmos, and hæmorrhage into the anterior chamber, while the ophthalmoscope revealed hyperæmia of the papilla and marked filling of the retinal veins. Yet Schoeler found that occlusion of the vortex veins gave only a very temporary increase of tension, and concluded that in living rabbits' eyes the value

of the vortex veins in carrying off fluid in high tension is small, and smaller in human eyes, in which two-thirds of the fluid leaves by the angle. To use his own words: "The tying of the vortex veins produces no remarkble increase of tension."

Kuehnt (7) reported finding in 15 glaucomatous eyes constant endoarteritis obliterans, signs of meso- and periarteritis, chiefly in the retina and ciliary body, and peri- and endo-phlebitis, which changes he thought were primary.

Birnbacher and Czermak (8) found chronic hyperplastic peri-phlebitis, with consecutive endo-phlebitis, producing narrowing of the vortex veins, which they considered might cause glaucoma. Later (9), they published an account of the condition of seven eyes enucleated for glaucoma of long standing. Their most remarkable observation was that of the peri-phlebitis and endo-phlebitis in the vortex veins, the latter probably a result of the former, and producing in some cases actual obliteration of the vessel. The changes in the vortex veins, the authors thought, could not be considered secondary to the raised intra-ocular tension, but they were secondary to an inflammation of the choroid, which was also present. They quoted Schultén to the effect that the ligature of one vortex vein may raise the intra-ocular pressure. The anterior chambers were also obliterated, and the discs were cupped. Birnbacher (10), in 1890, described very fully a case of glaucoma of only a week's duration, in which he found in the eye enucleated nine hours after death changes in the vortex veins, which he thought to be mainly responsible for the glaucoma, though the angle of the anterior chamber was closed up to its extreme periphery in one-third of the circumference, and in the remaining two-thirds the iris and cornea were in contact at a little distance from the periphery. The writer (11) has elsewhere discussed this case, and it will suffice here to say that Birnbacher found inflammatory changes in the walls of the vortex veins, which were, to his mind, chiefly responsible for the glaucoma.

Vossius (12) also related a case of glaucoma with periphlebitis and proliferation of endothelium, going so far as even to obliterate the four vortex veins. The angle of the anterior chamber was not closed.

Duerr and Schlegtenda (13), when examining five eyes

affected with hydrophthalmia, found indications of obstruction to the circulation through the vortex veins by pressure on the globe from the oblique muscles. (See Buphthalmia,)

V. Garnier (14) has seen in a vortex vein a peculiar condition of the inner surface narrow, tortuous, compressed, and due to the stretching and thinning of the sclera; also thickening of the intima, "compensatory to the pressure."

Knies (15) said that the periphlebitis and proliferating endothelium belong to late stages of glaucoma, and are not always present. From the supra-choroidea probably a considerable amount of fluid passes, which has not entered the vitreous through the lymph sheaths of the vessels, going to Tenon's capsule and lymphatics in the orbit. When the normal exits for fluid are interfered with much will leave the eye without entering the vitreous, and the internal membrane will then lose much nourishment.

Priestley Smith (16), who believed he had seen high tension induced through affection of the vortex veins in the early stages of suppurative tenonitis, examined two of these veins in each of 13 eyes lost by glaucoma—primary in 10 cases, secondary in 3—and, for comparison with these, the veins in 6 non-glaucomatous eyes, 3 of which were healthy in all respects. "In one case of subacute primary, and in one of secondary glaucoma the veins appeared to be partly obstructed by formations in the interior, such as have been described and figured by Czermak and Birnbacher. In several cases, both primary and secondary, the vein wall is perhaps thickened, and in some the perivascular space seems to be abolished by the close contact of the vein with the wall of the scleral channel which contains it. The close contact is present also in one of the healthy eyes. Regarded as a whole, the glaucomatous group presents, so far as my observation goes, no distinctive changes." He also suggests that any inflammatory changes which may be found in the veins would more probably be secondary than primary, especially considering the beneficial action in glaucoma of eserin and iridectomy.

Ulrich (17) examined the vortex veins in two cases of glaucoma absolutum (18), the remains of two eyes, glaucoma absolutum and glaucoma subacutum (19), one vortex vein in a case the history of which was unknown, and an eye with glaucoma hæmorrhagicum. Leaving out the

last, in none did he find the endo-phlebitis described by Birnbacher and Czermak, which Ulrich considered to be due to a hindrance in the vascular system of the uvea; but, like the inflammation of the angle, this endo-phlebitis should be looked upon as only a part of the general inflammation of the uvea. Still he thought that disease of the vortex veins might be the primary cause of a certain number of cases, and added that "further observations are necessary." The writer (20) has elsewhere published an account of the examination of the vortex veins of 20 eyes enucleated for primary glaucoma. The number of the vortex veins in eleven normal eyes was in one case 4, in three cases 5, and in seven cases 6, giving an average of 5.5, and they had a close relationship to the recti muscles, being generally near but very rarely beneath them. In the tabular statement of the condition of the choroid and veins "it will be seen that in eleven of the 20 eyes the choroid showed inflammatory patches, and that nothing abnormal was found in connection with the vortex veins except in three of the eyes examined. The choroidal inflammation always consisted of small but distinct aggregations of cells between the vessels, but not in the vessels themselves, not limited to any particular layer of the tissues, and never running so into one another as to give rise to the appearance of a change affecting it throughout. In no case was the lumen of any choroidal vessel seen to be diminished by exudaton pressing on it from without. The following is a short description of the pathological changes in the vortex veins of the three eyes above referred to. No. 13 shows a slight inflammation round the vein at its choroidal extremity. In this eye, as in some others examined, there were patches of inflammation in the ciliary body. No. 14 shows an inflamed patch in the wall and perivascular sheath of a vortex vein in the sclerotic, and the vein is partially filled by blood clot composed chiefly of red cells; in the wall of a vein just external to the sclerotic and close to the vortex vein (so that it might perhaps be a continuation of the latter) is a markedly inflamed patch. Inflamed patches are also found in the choroid and ciliary body. No. 16 shows patches of inflammation in the vein wall and perivascular sheath, and in the choroid; then in the lumen of the main vessel, but not in its wall, and not in any of the vessels of the choroid, are circular bodies taking on strongly the

hæmotoxylon nuclear stain. They are about one-fourth the size of a nucleus, give a bright reflex from the center, and are situated in patches in the red blood clot which occupies part of the lumen of the vessel. What these bodies are I am not at present prepared to say. They may, perhaps, be some form of parasite, but are too large for any of the known cocci, and what they may signify I have been unable to discover."

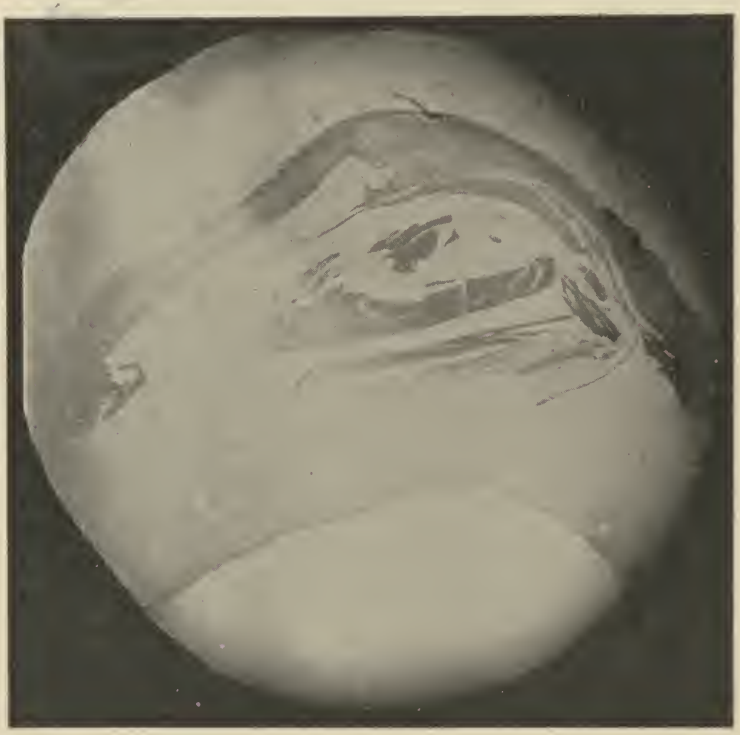

Fig. 6. Glaucoma in an Eye with Traumatic Aniridia. The whole Iris was knocked out through a wound in the Cornea: the Lens was wounded, and the Capsule adhered to the Corneal wound, resulting in a block of the ligamentum pectinatum and high tension.

In close connection with this subject is the **theory of Stellwag,** which is supported by Koenigstein (21), that the high tension, which is the important part in glaucoma, is due to augmentation of the vascular tension, general or local, assisted by a dilatation of the terminals of the arteries in the eye which prevents the easy access of the blood to the capillaries, and produces a stasis in the large capil-

laries and inflammation of the choroid; or, the increased intra-ocular pressure may be due to a narrowing or stoppage of the large vessels, especially the vortex veins, due to diminished elasticity and to shrinking in the sclera compressing them, faciliated by the obliquity of their passage from the eye, which is the theory supported also by Laqueur (22).

Jacobson's Theory

somewhat resembles the last. It is that glaucoma depends on a venous hyperæmia and stasis, especially in the anterior part of the choroid, due to diminished arterial pressure or senile degeneration of the vessels preventing the proper exit of the blood from the eye, with the result that Cloquet's canal and the vitreous "receive the products of the disease process," and high tension is set up.

In reviewing the work that has been done in connection with the vortex veins it must be admitted that insufficient authority has been produced upon which to construct a theory applicable to most cases of glaucoma, when one takes into account the results of ordinary treatment which, so far as we can see, can have only a very limited effect on such conditions of the vortex veins as have been brought forward as a cause of the disease, but which are really in all probability secondary changes. At the same time, it is evident that primary inflammatory affections may occur in these as in other veins, and that in such an event the subsequently swollen condition of the uvea would supply at least one of the abnormal conditions whose presence, even when insufficient of itself, might yet be a potent factor in the production of a cycle of events resulting in glaucoma.

Nicati's Theory.

Nicati's ideas (23) on the origin of glaucoma arise from his peculiar views concerning the structure and functions of the choroid. He holds that the choriocapillaris is contained in a closed sac, open only to the epithelium of the ciliary processes, and he has proved by experiment, to his own satisfaction, that the choriocapillaris participates, as a whole, in connection with the glandular epithelium extending from the root of tne iris to the ora serrata, in the elaboration of the aqueous. He holds besides, that this fluid is removed by the lymphatic

clefts and lacunar spaces of the iris, by the perivenous channels ending in the vortex veins, and by the anterior and posterior ciliary veins. Glaucoma can, therefore, arise from retention alone, due to (1) congenital insufficiency in the means of exit; (2) inflammatory engorgement of the iris; (3) progressive atrophy of the iris; (4) mydriatics by reducing the surface for absorption; (5) senile, rheumatic, and gouty changes in the iris. But another agent often comes into play in the shape of capillary varices associated with spasmodic œdema of the iris and choroid, the result being an excessive excretion of fluid within the globe. This may be set up by irritation of the iris, and should be considered as secondary to the first agent. Recent observations only tend to confirm the view advanced years ago, that the aqueous is secreted in the ciliary region, and Treacher Collins (24) has, by bleaching, exposed a series of glands hidden in the normal state by their own pigment, subject to catarrh, to cystic overgrowths, and to adenomatous tumour formation, and whose function he claims to be the secretion of this fluid. This theory has been accepted by many surgeons, while others have been unable to agree with Collins. John Griffith (25), for example, considering them too few in number for this important purpose, and to be more probably employed in controlling the amount of pigment in the eye, and in regenerating it as nature demands.

Greef (26) agreed with Nicati that the aqueous, non-coagulable when first drawn, resembles blood serum in its behavior after a second paracentesis, and the changes found in the ciliary processes in the eyes of rabbits removed at various intervals afterward sustained the idea that these originate the aqueous. John Griffith confirmed Greef's observations on the epithelium of the ciliary ridges, and like him found aqueous drops beneath it in newly excised eyes—"an exaggerated form of the normal secretory process." But, while most are agreed concerning the action of the ciliary processes, no confirmation appears to have been afforded of Nicati's observations on the chorio-capillaris, and Griffith denies all ground for the statement that the chorio-capillaris lies in a closed impermeable sac, and maintains that Nicati's assertion of a similiarity between its vascular arrangement and that of the ciliary processes is quite erroneous. He does not accept the ideas of either Collins or Nicati, but holds to

the old one of the ciliary excretion propounded by Young and again by Vetch (27) in 1820. Inflammation and œdema of the choroid have been discussed in connection with von Graefe's and Stellwag's theories. An inflammation of the chorio-capillaris was also supposed by Sattler (28) to have a relationship to glaucoma, but he has himself shown that it is absent in recent cases of simple glaucoma, while Klebs has found it to be only a secondary complication (Schnabel 29). We have already seen that the iris may be normal in glaucoma, and we know that it may be practically entirely absent without glaucoma, while the actions of operative measures and of miotics are difficult to reconcile with Nicati's theory of its causation.

Glaucoma Originating at the Optic Nerve.

This section should be read in connection with those dealing with papillitis and cupping of the disc, in which it is stated that certain authors believe that a morbid process is always present before the appearance of increased tension; while others, without going so far, consider that such a process is one of the earliest symptoms of glaucoma. It will be well to consider now whether sufficient evidence has been produced to justify a belief in disease in the region of the optic nerve as a proper cause of glaucoma, in the sense in which certain affections of the anterior part of the eye are so considered. Brailey (30), in 1877, said that no connection is shown, in his observations, between the optic disc, considered as a means of escape for the fluids of the eye, and increased tension; and again, that the optic nerve and its sheaths are not productive of glaucoma. In 1880 he (31) found "in most cases such a condition of the optic nerve as would hinder the passage of fluids along it, though this has not been proved by experiment to be a channel of exit, * * * and sometimes indications of an accumulation in the intergranule layer near the optic disc, and more often in the neighborhood of the ora serrata, besides * * * an accumulation in front of the lamina cribrosa and * * * sometimes * * a fissure or layer of loose tissue leading to this from the intergranule layer * * * consistent with the existence of a current from the outer retinal layers to the lymph channels of the optic nerve." Yet he added that "sclerosis of the optic nerve and lamina cribrosa when unaided has no effect on tension, for it exists with normal

tension in many cases of central disease." In 1881 he (32) noted that inflammation of the uvea, along with optic neuritis with slight haze and swelling of the papilla, is present at the commencement of primary glaucoma; and the same year, while asserting (33) that inflammation of the ciliary body, iris and optic nerve, is always present before increased tension, and that after recession of the ciliary muscle and dilatation of the circulus arteriosus major have occurred, a dense connective tissue forms between the fibres of the ciliary muscle and in the optic nerve, he still laid the blame for the glaucoma upon the anterior part of the eye.

Mauthner's theory (34) included a morbid process in the optic nerve, but did not suggest that glaucoma is caused by interference with filtration from the eye at that point; rather that the excavation is dependent upon a condition of the optic nerve which is not always present, and which, when it arises, is a result of choroiditis.

De Wecker (35) said in 1879, "that the idea *of an anterior and a posterior glaucoma*, which the younger Desmarres put forth some ten years ago, when he attributed the affection to inflammatory disturbance attacking two different circulatory systems, should perhaps, as Stilling proposes, be resuscitated for the channels of elimination." But one would require to know "how the vitreous humor * * * rids itself by filtration (through its central canal) of the products of elimination." De Wecker stated that he had always taught that the pericorneal region and that of the optic nerve are the only ones where the interior of the eye is without a hyaline amorphous membrane, quite unfit for filtration.

Schoeler (36), after experimenting on rabbits by tying the optic nerve and its sheath (he did not say whether in front of or behind the cenrtal artery), and injecting chloride of sodium in solution, concluded that "if the posterior lymph spaces (after Schwalbe) lie around the optic nerve and communicate with those in the retina and choroid, they are yet of no importance for removing fluid from the eye," and (37) "the assertion of certain authors that a glaucoma posticum can develop through hindrance in filtration from these spaces" is unnecessary as well as directly negatived by these experiments; also (38), in long continued and marked increase of tension caused by the injection of the solution into the vitreous it has never happened

that a flow could be discovered from the divided end of the optic nerve.

Stilling, who had drawn attention to the optic lymph path at the Ophthalmological Congress in 1877, again at the Heidelberg meeting in 1885, divided glaucoma into anterior and posterior, and stated that the optic nerve possesses an exit for the vitreous fluid. In nearly all glaucomatious eyes, especially in absolute and least in fulminating cases, he found dilatation of the dural sheath which he believed to be compensatory. By experiment he proved young eyes to fllter better than old, and those which were glaucomatous with the greatest difficulty. He concluded that glaucoma is a hypersecretion of normal fluid whose exit is hindered on account of senile discharges.

Ulrich (39) stated that only where the anterior part of the eye is inflamed does the optic nerve pass much fluid. Nettleship (40), in 1888, whilst noting the importance of any evidence pointing to the existence of a posterior glaucoma, and quoting cases of his own in which he considered that such might be its beginning, said that "the atrophic thinning, with little or no shrinking, of patches of the iris which is sometimes seen quite early in the disease," is one of the many evidences in favor of the belief that glaucoma, of all varieties of acuteness, usually starts in the ciliary region rather than in the optic nerve. Haensell (41), in 1890, considered that a hyaline degeneration of the optic nerve spreading to the vitreous is preliminary to the production of increased tension. Knies (42), in 1890, believed that in the beginning of glaucoma, and visible in all eyes before cupping has taken place, an infiltration of the optic nerve may be found, along with a similar condition in the corneo-sclera border, and the neighborhood of Schlemm's canal. Bitzos (43), in 1894, wrote that "one can accept as an absolutely certain fact that the glaucoma has begun by a papillitis," * * * and that a blocking of the lymph channel in the optic nerve accounts for all symptoms. But as ordinary optic neuritis does not render the lamina cribrosa more feeble, or produce increase of tension, we must suppose something peculiar about glaucomatous optic neuritis. The papillary lesion, constant in primary glaucoma, suffices to explain, by the hypertrophy of the connective tissue of the optic nerve and lamina cribrosa, which it causes, the obliteration of the optic path, if such exist. Galezowski (44), in 1894, be-

lieved that in simple glaucoma a spontaneous obliteration takes place in the lymphatics toward the back of the eye, and in the neighborhood of the optic nerve, whence arise the retraction of the lamina cribrosa, the hindrance to the lymphatic circulation in the papilla and the consecutive atrophy.

Knies (45) found signs of inflammation in the region of the sinus of the anterior chamber and optic nerve after introducing strongly irritating fluids into the vitreous, a condition which he had previously designated "glaucoma imminens," and, though the tension never rose and true glaucoma never appeared, he yet apparently here found confirmation of what he had already said of the common affection of the optic nerve in the stages of glaucoma before *plus* tension.

In connection with the above evidence it is not clear that interruption of a supposed optic nerve path would permanently close the anterior exits, and without that it is hard to understand how the tension could be permanently raised. Besides, it has not been settled that the optic nerve affords a passage for fluids of any great importance.

If it be true that optic neuritis is common at the commencement of glaucoma, it appears to be more likely that it occurs either as a complication of the true cause, or to be secondary to it, rather than that the neutritis itself originates the high tension.

Heredity and Constitutional States.

Gout has long had an evil reputation as a cause, or *the* cause of glaucoma, and even now, when a better knowledge of ocular pathology exists, gout is sometimes ranked among the obscure conditions of health which initiate the glaucomatous cycle. Beer is said to have pointed out the arthritic nature of glaucoma early in the present century. Sichel (46), in 1842, advised the use of anti-gout treatment for glaucoma, and in 1854 MacKenzie (47) said that the Germans considered glaucoma to be almost always due to gout, or gouty disease of the choroid. Hutchinson and Everbusch (48) thought that gout might be an important etiological factor in glaucoma, while Richey (49) is very strongly in favor of this view, and can find an explanation for every symptom on the hypothesis of a hyperplasia and subse-

quent contraction of the connective tissues of the eye. Walters (50) and others have reported cases benefited by anti-gout treatment, and Rabinowitsch has noticed asthenopia for near work disappear under the same treatment.

The above is sufficient to show how pertinaciously this idea has been held, and, perhaps, also how hypothetical its grounds. Modern criticism is not content without some fuller explanation of its *modus operandi*, and the effect of treatment based upon the theory, as well as its impotence in explaining many forms of the disease, do not justify the surgeon in laying great stress upon it as generally applicable.

Syphilis. I am unaware that any distinct proof has been produced that syphilis causes glaucoma, but there is an impression in the minds of some ophthalmic surgeons of the first rank that it is more common among those who have had, than among those who have not had, syphilis; and it seems very reasonable, when one considers the variety of the pathological products of this disease, and the frequency with which it attacks the uvea, that the initial process may be set up by it. Galezowski (51), for instance, has recorded two cases, one in a lady of 43 and the other in a child, in which iridectomy and mercurial inunction had a satisfactory effect, the probable syphilitic causation being the point to which he apparently wished to draw attention.

Rheumatism, also, but on what scientific grounds I am unaware, takes high rank with some ophthalmologists in this connection; so much so that they prescribe salicylate of soda as routine treatment in all cases of glaucoma.

The onset of glaucoma may be decided by exposure to cold (Nettleship 52), and besidas rheumatism, gout, syphilis, anæmia, and many *other constitutional diseases*, by "injuries of various kinds; cardiac, vascular and nervous disorders, neoplasms, congenital malformations, and senile changes. The primary, no less than the secondary, forms of the disease spring from various and complex causes, including many of those just mentioned. Primary glaucoma seems to depend sometimes chiefly on systemic, vascular or nerve disturbances, sometimes on individual peculiarity in the build of the eye, and often on a complex co-operation of several causes." (Priestley Smith 53.)

*Heredity** has long been acknowledged as an influence in the production of glaucoma, and that at an age earlier than usual, some local predisposition being generally blamed, the result of one or other of the diseases above mentioned, some other vascular or nervous affection, rigidity of the sclera, or the disproportionate size of the lens in a small globe. The last has the advantage of having been actually demonstrated, but it is not claimed by its author, Priestley Smith, that no other condition may be operative.

Races.—It has been frequently asserted that Jewish eyes are more liable than others to glaucoma, for instance, by Benedict of Breslau, in 1825, one-half of whose cases were Jews; by Knapp (60), and by Fuchs (61); but when considered in proportion to the whole population, writers have by no means unanimously adopted this opinion (62). The Egyptians, according to Brugsch Bey, and the negroes of Brazil, according to Moura (63), are especially liable to the disease.

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*NOTE.—The following are some of the published cases of apparently hereditary glaucoma: Von Graefe reported (54) families in which glaucoma appeared successively in several generations; Schmidt-Rimpler(55). a mother and two daughters, a father and two sons, a father and three sons, a mother and two sons; Mules (56), a father and two sons; Nettleship (57), who said that simple glaucoma is sometimes hereditary, and advised that "observations should be made on the discs of children of glaucomatous parents," reported a man with chronic glaucoma, whose daughter suffered from rainbows for some time, and whose son had large physiological cups, while a first cousin lost an eye from chronic glaucoma; Harlan (58), cases occurring for several generations in certain families; Somya (59), a father and three children, all myopic; and Arlt, Stellwag, Pagenstecher, Benedict, Mooren, etc., have recorded cases.

REFERENCES CONTAINED IN CHAPTER VII.

1. Leber—Arch. f. Ophth., XIX. 2, p. 145.
2. Weber—Von Graefe's Archiv.,, XXXIII, 1877, p. 81.
3. Schnabel—Arch. Ophth. N. Y., VII, 1878, pp. 12 and 243.
4. Priestley Smith—"Glaucoma," p. 36, quoting from Graefe-Saemisch Handbuch, II, p. 371.
5. Ditto, p. 28.
6 Schoeler—Reprint from v. Graefe's Archiv., XXV, 4, 1889, p. 48.
7. Kuent—Heidelberg Congress, 1885.
8. Birnbacher and Czermak—Arch. f. Ophth., XXX, 1885, p. 297.
9. Ditto, XXXII, 2, 1886, p. 1.
10. Birnbacher—"Ein Beitrag zuer Anatomie des Glaucoma Acutum," Graz, 1890.
11. Stirling—R. L. O. H. Rep. XIII, 4, 1893, p. 419.
12. Vossius—Lehrbuch der Augenh., 1888, p. 423.
13. Duerr and Schlegtenda—v. Graefe's Arch., XXXV, 2, p. 83, and Ophth. Rev., 1889, VIII, p. 339.
14. v. Garnier—Arch. f. Augenh., XXV, 1892, p. 24.
15. Knies—Centralbl. f. All. Path., April, 1890.
16. Priestley Smith—"Glaucoma," p. 129.
17. Ulrich—Arch. f. Augenh., XXVI, 1893, pp. 1, 19.
18. v. Graefe's Arch., XXVI, 3.
19. Ditto, XXX, 4.
20. Stirling—Op. Cit. (11).
21. Koenigstein—Wien. Med. Blaet., XV, 1892, p. 773.
22. Laqueur—v. Graefe's Arch., XXVI, p. 2.
23. Nicati—Arch. d'Ophth., Nov. and Dec., 1890, and Ophth. Rev. X, p. 106, and Mallassez in Central f. All. Path. 1890, I, p. 498.
24. Treacher Collins—Trans. Brit. Ophth. Soc., XI, March, 1891, and Lancet, Dec. 8, 1894.
25. Griffith—Brit. Med. Assn. meeting, 1894.
26. Greef—Arch. Ophth. N. Y., January, 1895.
27. Vetch—"A practical treatise on the Eye," London, 1820.
28. Sattler—Anzeiger d. K. K. Aertze in Wien., Dec.9, 1875.
29. Schnabel—Arch. Ophth. N. Y., VII, p. 23.
30. Brailey—R. L. O. H. Rep., IX, p. 199.
31. Brailey—R. L. O. H. Rep., X, p. 10.
32. Ditto, p. 275.
33. Ditto, p. 282.
34. Mauthner—Arch. Ophth. N. Y., VII, 1878, pp. 178, 242.
35. Wecker—"Ocular Therapeutics," 1879, p. 261.
36. Schoeler—Arch. f. Ophth., XXV, 4, p. 41.
37. Ditto, p. 44.
38. Ditto, p. 48.

39. Ulrich—Arch. f. Opht., XXX, 4, p. 235.
40. Nettleship—R. L. O. H. Rep., XII, p. 72, 215.
41. Haensell—Arch. d'Ophth., X, 1890, p. 518.
42. Knies—Centralbl. f. All. Path., April, 1890.
43. Bitzos—Annal. d'Ocul., CXII, p. 92.
44. Galezowski—Bull. de l'Acad. de Med. de Paris, XXXI, p. 240.
45. Knies—Arch. Ophth., XXIV, 2, 1895, p. 169.
46. Sichel—Annal. d'Ocul., VI, VII. VIII, 1841–42.
47. MacKenzie—"Diseases of the Eye," p. 899.
48. Everbusch—Die neue Ueniversitaets-Heilanstalt f. Augenh. Erlangen, p. 81.
49. Richey—Am. Jour. of Ophth. St. Louis, 1892, and Am. Jour. Med. Sci., CV, 1893, and Annals of Ophth. and Otol., V, 4, p. 920.
50. Walters—Klin. Monatsbl. f. Augenh., Jan., 1895, p. 1.
51. Galezowski—Receuil. d'Ophth. XIII, 1891, p. 654.
52. Nettleship—Op. Cit., (40), p. 215.
53. Priestley Smith—Ophth. Rev., XIII, 1894, p. 225.
54. v. Graefe—Graefe's Arch., XV, 3, p. 228.
55. Schmidt-Rimpler—Graefe-Saemisch Handbuch, V, p. 65.
56. Mules—Ophth. Rev., II, p. 49.
57. Nettleship—Op. Cit (40).
58. Harlan—Jour. Am. Med. Assn., V, p. 285.
59. Somya—Klin. Monatsbl. f. Augenh., XXXI, 1893, p. 393.
60. Knapp—Trans. Am. Ophth. Soc., 1888-89.
61. Fuchs—"Augenheilkunde," p. 384.
62. Graefe-Saemisch Handbuch, V, p. 66.
63. Moura—Trans. Inter- Med. Con. Washington, III, 1887. and Priestley Smith's "Glaucoma," p. 123.

CHAPTER VIII.

ETIOLOGY—(Continued).

Refraction and Glaucoma.

There is no doubt that the idea that hypermetropia predisposes to glaucoma has been fairly general in the profession, and with reason, for that has been frequently asserted by excellent authorities. For instance, Fuchs (1) says: "A disposition to inflammatory glaucoma appears to be present before all in the hypermetropic eye, whereas markedly short-sighted eyes are said to be little prone to the disease," which has been explained (2) as due to the fact that myopic eyes have deeper anterior chambers than emmetropic, and still deeper than hypermetropic eyes, while they have smaller ciliary muscles than either, and especially than hypermetropic, in which the association of a shallow anterior chamber and a large ciliary muscle, liable when swollen to press the iris against the cornea, is perilous to the health of the eye in certain circumstances. Stedman Bull (3) found in his cases of chronic glaucoma that hypermetropia was ten times more common than myopia. On the other hand, we must remember that hypermetropia is probably the commonest refractive state in most countries, and especially so in advanced age, as well as in childhood. Priestley Smith (4), whose cases, however, were not all chronic as Bull's were, found in 164 eyes hypermetropia in 45.1 per cent., emmetropia in 40.9 per cent., and myopia in 14.0 per cent.; and Kryonkoff (5) found hypermetropia in about the same proportion in cases of primary glaucoma, 43.18 per cent., emmetropia in 28 per cent. and myopia in 28.78 per cent.; while in more than 10,000 consecutive ophthalmic cases tested in regard to refraction he found nearly the same figures, hypermetropia 43.09 per cent., emmetropia 27.58 per cent., and myopia 29.32 per cent. This, though not absolutely conclusive, is strongly presumptive that glaucoma is not more frequent proportionately in hypermetropia than in other states of refraction. Nettleship (6), who like Bull, was considering chronic cases alone, found that in half his cases associated

with myopia the glaucoma began very early; in 18 cases only four had reached the age of 60; hence he concludes that myopia predisposes to glaucoma; or that both are due to one cause. Bull was not able to confirm Nettleship, for in 9 myopic cases occurring in his 90 cases, there were only two under 60 years of age.

Progressive Myopia and Glaucoma.

When high myopia is associated with glaucoma "the tension is rarely very high, though above normal, the field of vision becomes restricted in a manner more or less characteristic of glaucoma; and the papilla is excavated. It is the excavation of the papilla, as well as sometimes the unusual amount of pain complained of, which should lead one to suspect this complication. The excavation is never so deep as in other forms of glaucoma, but it is unmistakable whenever it is seen to reach out to the margin of the papilla. In these cases there is very often a very marked and excessive degree of choroidal atrophy surrounding the papilla, and it is generally supposed to be owing to the comparative want of resistance of the surrounding parts that the yielding of the lamina cribrosa does not assert itself to the same extent as would otherwise be the case" (Berry 7). Fuchs (8) said that "choroiditis and high myopia frequently give rise to increased tension under the picture of simple glaucoma." Noyes (9) remarked that in "myopia glaucoma is hard to diagnose." One idea, illustrated from the above quotation from Fuchs, is that the myopia causes the glaucoma, which is supported by Berry (10), who wrote, "glaucoma in a highly myopic eye is, properly speaking, secondary inasmuch as the choroidal changes which lead to the elongation of the globe probably interfere at the same time with the circulation in that membrane and lead to a glaucomatous complication;" and Knies (11) remarked that passing high tension is found in progressive myopia. Somya (12) related four cases in one family where the father aged 63 and three children aged respectively 26, 15, and 13 years, all suffered from high myopia and glaucoma, but he knows "no reason why scleractesia posterior should lead to glaucoma," and it appeared to him "more probable that the myopia on one side, and the glaucoma on the other, had been inherited, without the one originating or aiding the development of the other." Again in 1881, Brailey (13) wrote that in children

owing to the comparative elasticity of the globe "the increasing contents of the vitreous chamber do not require to press so much upon the lens, which in its turn has greater power of resistance owing to the greater tension of the zonule. Thus, the dangers attendant on the contact of iris and cornea are frequently averted. Such cases often present themselves, in my opinion, as cases of progressive myopia." Nettleship (14), who considered the combination of myopia and glaucoma to necessitate a bad prognosis, expressed his belief in 1888, as already stated, that either myopia predisposes to glaucoma or that both are due to the same local cause.

Of these three ideas it is hard to say which is the correct one, or whether each has not its measure of truth. But it seems easy to understand how in eyes only slightly predisposed to posterior staphyloma—on account of local weakness in the sclera, dragging from shortness of the optic nerve, or otherwise, an increased intra-ocular tension might be sufficient to turn the scale and initiate myopic bulging. The following case (15), a *multum in parvo*, is interesting in this connection. Rose S. aet. 18, *R. E.* T. + 1. M. 12 D. Cornea — 9 m.m. in diameter. Iris absent, except a narrow crescentic piece on the inner side for $\frac{2}{5}$ of a circle. Lens in situ and with a diameter equal to that of the cornea O. D. cupped. Myopic crescent on outer side. Opaque nerve fibres. *L. E.* T. + 2. M. and cornea as in *R. E.* No iris except three small isolated bits on the inner side. O. D. cupped. Here we have eyes strongly predisposed, according to Priestley Smith's investigations, to primary glaucoma—very small corneæ, lenses disproportionately large for the diameter of the eyes in the region of the ciliary bodies; probably, at least at one time, small in all directions, and possibly even hypermetropic.

The theory of myopic choroiditis spreading forward does not seem to commend itself as in accord with clinical experience, while those who uphold a "posterior glaucoma" might find satisfaction in explaining the high tension as secondary to an interference with the health of the eye in the region of the optic nerve.

Effects of Accommodation.

MacKenzie (16) considered overuse of the accommodation to be a cause of the "choroiditis of glaucoma;" and

Walker (16, a.) thought that a long continued overexertion of the accommodation produces an irritable state "which only wants some provocation (such as atropin) to develop an active inflammation." W. Schoen (17) laid special stress upon two points: He believed (1) that the lamellæ of the suprachoroidea are the tendons of the central fibres of the ciliary muscle and proceed to the sheath of the optic nerve; and (2) that the zonule anatomically falls into an anterior and a posterior division which have different functions in accommodation. Schoen says that the various forms of glaucoma are due to the actions of the different divisions of this muscle, and that normally the sclera is protected from intra-ocular pressure by the above mentioned tendons, which also by dragging on the optic nerve produce the glaucomatous cupping. "In glaucoma simplex all the muscle fibres are active, and the cup is the result of the dragging of the fibres on the optic nerve. In glaucoma acutum the inner and ring fibres are quiescent, while the external meridional still contract and retain their tone. In glaucoma atonicum all the fibres refuse work." Snellen (18) interprets Schoen's ideas as a hypothesis "that after a strain of accommodation the ciliary muscle becomes suddenly relaxed, and that as a result of this the lens is suddenly pressed forward," and certainly there appears to be little ground for regarding it as anything more than a hypothesis, while it is difficult to understand how relaxation of the ciliary muscle should result in propulsion of the lens.

Quite recently (18 a) Schoen has asserted that if only every one who requires glasses would wear them in good time there would be no more glaucoma; of his last 140 cases 48 per cent. were hypermetropic, and not one of them wore a distance glass; 33 per cent. were astigmatic, always uncorrected; 20 per cent. wore no glasses at all, or lenses which were unsuitable. Women shrink from wearing glasses, and therefore, produce twice as many glaucoma patients as men do.

Snellen himself (19) gave a much more satisfactory explanation of the effects of accommodation in the production of glaucoma, when he observed that the diameter of the lens does not in age, as it does in youth, diminish *pari passu* with the increased diameter of the ciliary muscle on its contraction, whereby the perilenticular space remains nearly constant in width; but that, on the contrary, while

the muscle continues on accommodation to advance toward the lens, the sclerosed condition of the latter prevents its retreat to a corresponding degree, the two coming possibly into firm contact, while the slackened zonule permits of the advance of the lens. Snellen finds confirmation of this in some of Priestley Smith's investigations.

Priestley Smith (20) remarked that the connection between strain of accommodation and glaucoma can by no means be denied, but that "it must be remembered that the liability to glaucoma is greatest at a time of life when the accommodation is in abeyance. Much book-work is likely to be injurious, apart from the accommodative act, by reason of the stooping and congestion of the head which it often involves." In a case such as that related by Noyes (21) of an ophthalmic surgeon who, being astigmatic and also glaucomatous, stops the glaucoma by suspending accommodation with atropin and the use of full correction, it is improbable that a primary interference with excretion takes place at the angle of the anterior chamber, and most probable that it originates in connection with the lens and ciliary body in some such way as that described by Snellen and Priestley Smith.

While considering the connection between accommodation and glaucoma it will be interesting to compare the former with the action of the most potent medicinal cure for glaucoma, eserine. During accommodation we have convergence of the optic axis, (which may be left out of consideration here), diminution in the size of the pupil, and contraction of the ciliary muscle; during the action of eserine we find a similiar contraction of the pupil, as well as action of the ciliary muscle productive of myopia. As eserine benefits glaucoma, why should not accommodation? The difference between their actions is probably chiefly one of degree, the eserine contracting the pupil very markedly, the reason generally given for its salutory effects, and possibly also producing a state of greater hyperæmia of the iris, which perhaps should not be altogether ignored when taken in conjunction with its probable effects upon a previously congested ciliary body, while the ciliary muscle is thrown into a state of prolonged spasm. It has been said by excellent authority that cases of glaucoma have actually been produced by eserine, and it is not an uncommon experience that both eserine and pilocarpine act not only without benefit, but appear dis-

tinctly to accentuate the disease. Are some of these cases in which, from the inelastic condition of the lens or the laxity of the zonule, excessive accommodation might have had a similiar effect?

Glaucoma Caused by Mydriatics.

MacKenzie (22) found it necessary to relinquish the use of atropine in certain cases of glaucoma on account of an increase of symptoms due to it; and (Laqueur 23) in 1877 drew the attention of the profession to the effects of calabar bean and atropine in this disease, and cited experiments made by others on the intra-ocular tension under their influence.

It had long been understood that atropine reduced the tension in corneal and iritic diseases; and Pflueger (24) found in 81 per cent. of all eyes distinct diminution, in 16 per cent. no change, and in 3 per cent. increase of tension on account of its local application. Adamuek (25) confirmed the observations of Wegner, Gruenhagen, and Coccius on animals, that it lessened the tension. Wharton Jones (26) said atropine was dangerous in acute glaucoma, and v. Graefe (27) had noticed a "latent chronic glaucoma" changed into an acute attack by atropine. Laqueur believed that though it is a means of increasing tension "this action does not appear so long as the circulatory apparatus is in good working order." G. E. Walker (28) considered it a typical provocative of glaucoma, "since it excites the radial fibres to drag on the congested and irritated circular muscle." Story (29) related a case of glaucoma in one eye and a month later in the other, appearing suddenly in each case two hours after the subconjunctival injection of cocain for operations on the external muscles. Walter (30) published two cases of glaucoma produced by scopolamine, which was on its first introduction considered by some surgeons, without very obvious reason, as a safe mydriatic as regards glaucoma; and other cases have been reported.

The above is sufficiently illustrative, and it may with safety be said that without exception all mydriatics are to be avoided in an eye predisposed to glaucoma, and that their action should be watched in every case, and more especially after middle life.

The most generally accepted explanation of this, and that which has the firmest scientific basis, lies in the fact

that mydriatics crowd the iris into the filtration angle, and tend to block it. Still it is as well to bear in mind their paralyzing effect on the ciliary muscle, as well as the possible hyperæmia of the ciliary body produced by the emptying of the vessels of the iris on its dilatation, mentioned by Fuchs (31) as the probable reason for the ill effects of atropine in some cases of irido-cyclitis. Knies (32), to support his theory that glaucoma is due to an irritation of the anterior exits for the intra-ocular fluid caused by a glaucomatous poison circulating in the eye, supposed that mydriatics, causing hyperæmia of the fundus, accelerate tissue metamorphosis, "so that the nocuous elements contained in the latter are hurried along faster and in greater quantity toward the channels of outflow of the eye."

Glaucoma Caused by Operation.

It has long been acknowledged that an attack of glaucoma in the previously sound eye may be precipitated by an operation on the other, so much so that it has become a very general proceeding to instil eserine into it beforehand, as was at first recommended by Arlt. The frequent depression of mind and body produced by the circumstances has been very greatly credited as the cause, and Fuchs (33) found in this two probable factors, the interference with the circulation, and the resulting reflex dilatation of the pupil. Swanzy (34) thought that the dilatation of the pupil, the immediate cause, is a consequence of confinement in a dark room.

Ocular Hyaline Degeneration and Glaucoma.

A hyaline degeneration of tissues has been proposed as the foundation for the glaucoma process and somewhat elaborated by Haensel. Vossius (35) in 1888 related a case in which hyaline degeneration and sclerosis of arteries in the choroid, and cellular infiltration round the veins, co-existed with obliteration of the angle of the anterior chamber; and Klebs (36) in 1889 found hyaline degeneration in the vessels in a number of cases of blind glaucomatous eyes, and in one recent case. He thought this had a close casual relationship with glaucoma. V. Garnier (37) in 1892 was convinced that there is a diminution of the natural elasticity in the choroidal vessels when spontaneous increase of tension is present. Haensell (38) said that the cellular matter which in fœtal life unites the

vitreous lamellæ arranged around the branches of the hyaloid artery, transforming later so as to leave the vitreous like jelly, in glaucoma condenses so that the passages are completely closed to the circulating lymph, an accumulation takes place between the lamellæ which augments the intra-ocular tension, and from lack of proper nourishment the vitreous atrophies. Other parts of the eye show analagous changes, and to this interference with nutrition Haensell ascribed glaucoma, beginning sometimes with softening and excavation of the optic nerve, the increased tension setting in only after the degeneration has spread thence to the vitreous. On the other hand, when the vitreous is first affected tension may arise and continue without cupping, and even with normal visual acuity. Priestley Smith (39) admitted as a possible hypothetical cause of some cases changes in the hyaloid membrane, in the stroma of the vitreous, or in the constitution of the vitreous fluid; while Knies (40) has found the vitreous generally normal except in the late stages to which it and the hyaline degeneration of vessels, when present, belong. In the glaucomatous eyes whose arteries one has most opportunity of examining the vitreous is frequently found to be much shrunken and detached, especially posteriorly.

Sclerosis of Tissues and Glaucoma.

It is generally admitted that the sclera is more rigid in age than it is in youth, but some writers assert that it is specially so in glaucoma, which may result from this inelasticity. Gosetti (41) and Del Monti (42) are quoted by Knies as stating, the former that glaucoma is caused by a general sclerosis of the ocular tissues, and the latter that it may be due to a sclerosis of the anterior part of the sclera and of the cornea. Richey (43) is able to find an explanation for every feature of glaucoma on the hypothesis of a general sclerosis. Arlt (44) believed that special rigidity of the sclera causes stasis, the first stage in glaucoma; and Straub (45) noticed less elastic retraction of the choroid from the sclera on incision than in normal eyes. Schmidt-Rimpler (46) considered rigidity and alteration in the elasticty of the choroid to be important, as did Weber (47), Koenigstein (48), v. Garnier (49), and others; whilst Somya (50) suggested that in hereditary glaucoma the fault might lie in rigidity of the sclera. On the other hand, Hulke, Nettleship, Schnabel (51), and

Brailey (52) have found no special rigidity in glaucoma.

Glaucoma Caused by Changes in the Shape of the Globe.

Roeder (53) thought that a tendency in advanced age for the eye to become cone-shaped inclined to narrow the angle of the anterior chamber, drag on the ciliary processes, and produce an excessive excretion from them. Von Garnier (54) considered that when scleral stretching exists the iris and cornea are brought into apposition, and the angle is closed. It is well known that staphyloma of the cornea and of the ciliary region are not infrequently associated with high tension. They are sometimes caused by the glaucoma, and sometimes the altered relations of the cornea and iris interfere with the excretion of ocular fluid and produce it. In exceptional cases glaucoma may arise in interstitial keratitis, with or without bulging of the cornea, and independent of synechia, or in softening of the cornea associated with pannus. In some of these cases frequent sclerotomies have a most beneficial effect.

REFERENCES CONTAINED IN CHAPTER VIII.

1. Fuchs—"Lehrbuch der Augenheilkunde," p. 384.
2. Ditto, p. 718.
3. Stedman Bull—Trans. Am. Ophth. Soc., 1889.
4. Priestley Smith—"Glaucoma," p. 122.
5. Kryonkoff—Quoted by Priestley Smith, Op. Cit., p. 123.
6. Nettleship—R. L. O. H. Rep., XII, pp. 72 and 215.
7. Berry—"Diseases of the Eye," p. 218.
8. Fuchs—Op. Cit., p. 406.
9. Noyes—In Gruening's paper, Trans. Am. Med. Soc., 1889.
10. Berry—Loc. Cit. (7).
11. Knies—Centralbl. f. Allgem. Path., April, 1890.
12. Somya—Klin. Monatsbl. f. Angenh. XXXI, 1893, p. 390.
13. Brailey—R. L. O. H. Rep., X, p. 372.
14. Nettleship—Loc. Cit. (6).
15. Brailey—Trans. Ophth. Soc. of the United Kingdom, 1890, p. 139.
16. MacKenzie—"Diseases of the Eye," 1854, p. 571.

16a. Walker—"Essays on Ophthalmology," 1879, p. 63.

17. W. Schoen—Graefe's Arch. 1887, 1-2, p. 195.
18. Snellen—Ophth. Rev. Lond., X, 1891, p. 47.

18a. Schoen—Wien. Klin. Rundsch. No. 26-31.

19. Snellen—Seventh International Congress, Heidelberg, 1888.
20. Priestley Smith—"Glaucoma," p. 131.
21. Noyes—Loc. Cit. (9).
22. MacKenzie—Op. Cit. (16).

23. Laqueur—Arch. f. Ophth., XXIII 3, p. 149.
24. Pflueger—Arch. f. Augen. and Ohren., II, 1.
25. Adamuek—Annal d'Ocul., LXIII, p. 108.
26. Wharton Jones—Med. Times and Gaz., I, p. 111.
27. Graefe—Archiv. XIV, 2, p. 117.
28. Walker—Op. Cit. (16a), p. 63.
29. Story—Ophth. Rev., XII, 1893, p. 6.
30. Walter—Klin. Monatsbl. f. Augenh., Jan., 1895, p. 1.
31. Fuchs—Op. Cit. p. 332.
32. Knies—Arch. Ophth., XXIV, 2, 1895, p. 185.
33. Fuchs—Op. Cit. (31).
34. Swanzy—"Diseases of the Eye," p. 312.
35. Vossius—Lehrbuch der Augenh.," p. 423.
36. Klebs—Arch. f. Allgem. Path. II.
37. Von Garnier—Arch. f. Augenh., XXV, 1892, p. 24.
38. Haensell—Arch. d'Ophth., X, 1890, p. 518.
39. Priestley Smith—"Glaucoma," p. 130.
40. Knies—Op. Cit. (11).
41. Gosetti—Annali di Ottalmol., V.
42. Del Monti—Annali di Ottalmol. VI, p. 662.
43. Richey—Amer. Jour. of Ophth., 1892.
44. Arlt—"Zur Lehre vom Glaucom," 1884.
45. Straub—Arch. f. Ophth., XXXIV, 3, 1889, p. 195.
46. Schmidt-Rimpler—Graefe-Saemisch Handbuch, 1875.
47. Weber—Arch. f. Ophth. XXIII, 1877, p. 1.
48. Koenigstein—Wien Med. Blaet., XV, 1892, pp. 773, 789, 809, XVI, pp. 17, 35, 51.
49. Von Garnier—Loc. Cit. (37).
50. Somya—Loc. Cit. (12).
51. Schnabel—Arch. Ophth. N. Y., VII, 1878, pp. 12 and 243.
52. Brailey—R. L. O. H. Rep., IX, 1877, p. 199.
53. Roeder—Arch. f. Augenh., 1871, IX, p. 164.
54. Von Garnier—Loc. Cti. (37).

CHAPTER IX.

GLAUCOMA IN THE YOUNG; IN ANIRIDIA AND COLOBOMA OF THE IRIS; AND IN APHAKIA.

Glaucoma in the Young. Glaucoma in young people may be divided into two groups, that constituted by one or other of the forms of "*senile*" *glaucoma, only occurring at an unusually early age*, and with nothing else specially to separate it from ordinary glaucoma; and that in which an abnormal dilatation of the globe, and especially of the cornea, results from the excess of pent-up fluid in the interior of an undeveloped and distensible eye, a disease to which the names *buphthalmia*, *hydrophthalmia* and *cornea globosa**, have been given. The essential difference between buphthalmia and ordinary glaucoma lies, therefore, not in the causation of the increased tension, but in the less power of the globe to retain its natural dimensions under pressure, care being taken to distinguish the equable dilatation of the whole eye from a mere staphyloma.

I. **Buphthalmia.**—*Symptoms.* Superfluous intra-ocular pressure, whether as a result of some invisible cause and, therefore, called primary, or secondary to, for instance, a leucoma adherens, not being met by such a resisting capsule as in an older eye, is able to distend the globe by dilatation and thinning of its walls to a diameter of even 38 m.m. from before backward, and 28 m.m. across. In a case of Warlomont's the cornea was 17 m.m. in diameter. The effect of this is to let the dark uvea shine through the sclera, to enlarge and sometimes flatten the cornea, which may be clear or opaque; and, as the lens does not participate in the abnormal enlargement, the zonule, on account of the separation of the elongated ciliary body from the lens, is stretched, maybe ruptured, with luxation of the lens which, in any case, is flattened, sometimes

*Note.—Schweigger (1) asserts that "cornea globosa" should be applied only to cases of abnormally large cornea with deep anterior chamber, and without stretching or thinning of the sclera, but does not explain the origin of such cases, and the above terms are used indiscriminately by others (*e, g.*, Fuchs and Swanzy).

opaque, and changed into a spongy network with thickened capsule, or occasionally is absorbed. This lack of support behind produces irido-donesis, the anterior chamber is deepened, while myopia necessarily accompanies the antero-posterior elongation of the globe to an extent which must be modified by the state of the lens. The ciliary body and choroid may atrophy. The disc is frequently cupped in spite of the general yielding of the walls, which may, however, render less apparent the tendency to increase of tension. The retina is sometimes detached, secondarily to the detachment of the hyaloid from the retina, owing to the fact that the vitreous may not expand to the same extent as the globe.

The enlargement of the globe may cause an increase in the size of the orbital cavity. The vision is poor, the field contracted, and photopsia and nystagmus are sometimes found. These symptoms, though they may remain stationary, or even diminish, are apt to progress toward greater destruction of the ocular functions.

Etiology. Cases of buphthalmia may be divided into those which are secondary to some visible pre-existing cause, and have their counterpart in the secondary glaucoma of adults, and those which are congenital.

(a) **Secondary Buphthalmia** is by far the commoner, and cases have been published within recent times by—among others—Brailey (2), two cases with posterior synechia and small immovable pupil, T +, and optic disc cupped; and five cases (3) in which the iris was partly, or quite, adherent to the cornea; and by Treacher Collins (4), five cases, four due to adhesion of the iris to a perforated cornea, and the fifth to intra-ocular tumor. The average antero-posterior diameter of those globes, the measurements of which are given, is nearly 30 m.m., the vertical about 25 m.m., while the corneal was about 14 m.m. A globe has been examined which measured 38 m.m. from before backward, and 28 m.m. across (Schiess 5). In each of Collins' cases the disc was cupped.

(b) **Congenital Cases.** Cross, of Bristol, has reported a case in which the antero-posterior diameter of the globe reached 40 m.m., and another which measured 28 m.m. across, and whose cornea was 19 m.m. in diameter.

Various theories have been advanced to account for these cases, which are usually binocular, and which have been found in the pig and horse, as well as in man; as,

for instance, that they are due to an arrest of development of the cornea before differentiation takes place between it and the sclera (Steffan 6, Mueller 7). Duerr and Schlegtenda suggest that the oblique muscles by pressure indent the sclera and interfere with the circulation through the vortex veins (8). They describe five cases, the important points in Case I, a girl aged 18, being the presence of round-celled infiltration at the cornea-scleral junction, with many vessels, some entering the cornea; and that Schlemm's canal was lost to view except in a few sections, where it was just visible, while the angle was open. The disc was cupped. Case II—No history; had a similar, though less marked, condition of the corneo-scleral junction. The angle was occluded in places, lens shrunken and calcareous, Schlemm's canal obliterated in places by round, spindle and pigment cells: optic disc cupped, vortex veins normal. Case III—Boy, aged 12, eyes affected since infancy, cornea infiltrated and vascular, two vortex veins had both trunks and branches pigmented externally, and one was contracted in caliber; optic disc cupped. Case IV—Boy, aged 19, eyes gradually enlarged from infancy; left eye had a cornea measuring 16.5 m.m. vertically, and the globe was 35 m.m. from before back and 27 m.m. across. Cornea was vascular, angle open, no canal of Schlemm, and spaces of Fontana partially filled by cell infiltration; optic nerve degenerated; the vortex veins were dilated under slight staphyloma of the sclera. Case V., the right eye of Case IV. In the line of the superior oblique was a deep groove; there was no trace of Schlemm's canal or of the spaces of Fontana. The vortex veins did not indicate retarded circulation so plainly as in Case IV., though that was sufficiently evident.

On reading these cases one can hardly help concluding that there is too little foundation for the belief that any one of them was produced by muscular action, for, except perhaps in Case III., there is ample reason in the condition of the corneo-scleral junction and the angle for the production of increased tension, and it is only to be expected that in an enlarging and soft globe contracting muscles encircling it should leave their mark as a constriction.

Other authors (Horner and Meiralt in Zurich, 1869, Gallenga, Raab (9), Mauthner (10), Pflueger (11), Dufour (12), consider buphthalmia to be due to pathological

causes, such as intra-uterine irido-choroiditis, or to congenital weakness in the zonule of Zinn and corneo-scleral ring; while Panas (13) adds that the type is that of consecutive glaucoma, due to the frequent occlusion of the iritic angle by excess of secretion.

Buphthalmia is sometimes hereditary, as in Randolph's case (14), where a brother and sister were affected; the first of Duerr and Schlegtenda's cases, above quoted, where the parents were cousins, and two of the patient's brothers, aged respectively 20 and 8 years, suffered in the same way; and one case noted by Treacher Collins (15) whose sister suffered like herself from hydrophthalmia. Warlomont (16) published a case in a patient aged 13 with a cornea measuring 17 m.m. across, and with a vision of $\frac{2}{3}$. Treacher Collins (17) described five eyes excised for primary buphthalmia, and asserted his belief that congenital cases are probable "due to some defect of development in the filtration area at the angle of the anterior chamber. In some cases it appears that the canal of Schlemm (Case II.) is absent; in others, that the iris does not become separated from the back of the cornea up to its extreme periphery, and that a congenital adhesion in this position prevents the exit of the fluid from the eyes." Later, Collins brought forward a new theory (18), founded upon developmental physiology, which has received the support of Priestley Smith, Cross of Bristol and others, and which certainly appears to be well founded. Collins referred to the facts that before the appearance of the iris in the fœtal eye that portion of the epiblast which goes to form the lens is cut off from the cuticular epiblast by the ingrowth of the mesoblast, which is divided into two layers, the posterior forming the anterior fibro-vascular sheath, and the anterior forming the substantia propria of the cornea and Descemet's membrane. At this time there is no anterior chamber, and the lens is separated from the cornea by the anterior fibro-vascular sheath alone, which is in contact with both. As the iris grows out from the ciliary body, carrying in front of it the prolongation of the posterior fibro-vascular sheath, which goes to join the anterior, it insinuates itself beneath the anterior sheath and raises it up from the surface of the lens. The sheath ultimately becomes united to the iris, whose anterior layer it forms, while that portion filling the pupil beyond the limit of extension of the iris takes the name of "pupil-

lary membrane," which ought to disappear, but occasionally remains in part persistent, and may be seen proceeding from the corona of the iris to the lens capsule, and even to the back of the cornea. When the aqueous begins to be secreted and the lens to be flattened the cornea and iris separate, unless from failure of the normal complete absorption of the bands between them. It is to the pressure of such corneo-iritic adhesions or anterior synechia that Treacher Collins ascribes at least some cases of congenital hydrophthalmia. In cases recorded by Beck (19), Samelsohn (20), Makroki (21), and Zinn (22), there existed some doubt whether intra-uterine corneal perforation might not have caused the synechia, but Collins described a case in which the cornea is without a sign of such perforation, but a fibrocellular membrane remained behind the lens, and a large piece of a persistent pupillary membrane, as well as the pupillary border, attached the iris to the cornea. Collins has seen other cases of the kind, one adhesion passed between the iris and the ligamentum pectinatum. In the next the adhesions were situated rather nearer the periphery of the iris than its pupillary border in its outer half, were 12 in number, and appeared as little whitish elevations of the iris tissue coming forward to the back of the cornea, which latter was perfectly clear. This eye also contained some slit-like openings through the substance of the iris just external to its sphincter muscle. In another the iris was adherent at its upper part to a clear cornea, and at the periphery closely attached to the cornea on both sides of the section. Posterior synechia also existed, but no round-celled infiltration in any part of the uvea. The anterior fibro-vascular sheath in this case appears to have been unable to separate from the cornea, both in this region and at the periphery. In another case Collins found extensive peripheral adhesion of the root of the iris to the cornea, and in several other eyes affected with congenital glaucoma, and in which iridectomy had been unsuccessfully performed, he found with the microscope in each one that the angle of the anterior chamber was closed in the region of the coloboma, as well as elsewhere.

In the transactions of the British Ophthalmological Society, 1895-6, Cross of Bristol has detailed an examination of three eyes excised for congenital buphthalmia. They all

had deep anterior chambers; the first showed definite adhesion of the root of the iris to the ligamentum pectinatum: the second, no definite adhesion, but strands of tissue suggestive of a former contact of iris and cornea, which had given way; and the third, a distinct block of considerable width, occluding the angle. Cross concludes that his "specimens are in strong support of the view that the essential cause of congenital hydrophthalmos lies in the imperfect condition of the filtration angle." Devereux Marshall at the same time showed a hydrophthalmic eye in which the anterior chamber was very deep, and yet its angle was distinctly closed.

How is it that such cases ever improve with advancing age? An explanation may lie in the condition found in an eye examined by Collins, which had to be removed on account of a blow. In it fine adhesions uniting iris and ligamentum pectinatum appeared to have been stretched until the angle had become widely open.

II. The second group is that which includes cases of **ordinary primary glaucoma without dilatation of the globe, but occurring at an unusually early age.** These cannot properly be separated from "senile" glaucoma, and are introduced here chiefly to distinguish them from buphthalmia, and to illustrate the ages at which increased intra-ocular tension may exist without causing enlargement of the eye. For instance, in recent years Nettleship (23) mentions 15 cases beginning before the age of 30 years. In nearly two-thirds the eyes were myopic; 11 of these were between 20 and 30, and the remaining four were under 20 years of age. Both eyes were affected in seven of the cases, and of the four cases below 20 years old both eyes were affected in only one. Iridectomy was beneficial, even though two of the cases operated on had considerable myopia. "It is worth noting that in one case of double glaucoma at about 20, with very high myopia, the patient, a male, was almost a dwarf and obviously cretinous, and that in three others, all girls, the patients were stunted, feeble and backward." Spencer Watson (24) published the history of a case in a woman, aged 23, and Brailey (25) a case of a girl aged 18, with in the right eye myopia of 12 D., opaque nerve fibers, and a cornea of 9 m.m. in diameter. T+1. The left eye was also myopic; cornea measured 9 m.m. T + 2. The optic nerve was cupped, and the iris

was nearly entirely absent in both eyes. Priestley Smith (26) found that among 1,000 cases of primary glaucoma the average number beginning under the 20th year is less than 1 per cent., and that the increase with advancing age is slow for a considerable time longer, the average being a little higher in the decade between 20 and 30. I have already quoted an interesting case (see Glaucoma Produced by Mydriatics), published by Story (27) of a girl aged 13 in whom acute glaucoma appeared in

Fig. 7. Glaucoma in an Eye with Apparent Congenital Aniridia. A congenital adhesion unites the root of the Iris to the Cornea, due to an insufficient development of the normal separation of the Iris and Cornea. There was also anterior polar cataract. This patient's mother, who had the same condition of the Iris, acquired Glaucoma secondarily to a corneal ulcer.

the right eye after the use of atropine and cocain, and an operation on an external ocular muscle of that eye, and a month later in the left eye after cocain and a similar operation upon it. In the same paper Story relates the case of a girl of 18, with a cornea of a diameter of 12.5 m.m., deep cups and T + 2, and quotes the following from Schmidt-Rimpler in the Graefe-Saemisch *Handbuch:* Glaucoma in a girl aged 9 (Stellwag), a boy of 12 (Schirmer), a boy of 16 (Mooren), a boy of 19 (Mooren), a woman of 20 (Peffmueller), and a

"patient" of 22 (Schirmer). Many other cases have been observed in young people, and the above are given merely as examples.

In Aniridia and Coloboma of the Iris.

That glaucoma could occur in an eye in which no iris is visible by external inspection seemed to clearly prove that any theory in which it took a principal part must be erroneous; and such cases have been recorded by Windsor (28) of Manchester, H. Pagenstecher (29), Armaignac (30), Brailey (31), Lang (32), De Schweinitz (33), Rindfleisch (34), etc., while Treacher Collins (35), besides describing three cases, discussed the etiology of both the glaucoma and the conditions to which it is due. It is the rule to find that where there is apparently none there is really an undeveloped iris hiding behind the corneoscleral junction, its meager extent being probably due, as suggested by Manz, to a pathologically prolonged continuance of the fœtal contact of the lens and iris, preventing the ingrowth of the latter. But the narrow iris is theoretically of sufficient width to block the filtration angle, and practically this is found to have taken place in some cases at least by means of congenital bands passing between iris and ligamentum pectinatum. The argument of these cases against the angular retention theory is, therefore, found to be invalid. (See Congenital Buphthalmia.) In traumatic aniridia glaucoma may supervene on adhesion to the corneal wound of the lens capsule, ciliary processes, or vitreous.

Glaucoma in Aphakia.

Considerable importance has been attached to the fact that glaucoma may appear in the aphakic eye, because this has been assumed by a number of writers as sufficient evidence of the error of such views as ascribe an important place to the lens in the etiology of increased tension. That such assumption was premature, and lacking in sound foundation, will immediately appear. Bowman (36) in 1867, and v. Graefe (37) in 1869, had observed glaucoma not uncommonly after extraction, the former noting a special liability to it after needle operations, and not due to iritis, while the latter blamed an irritation of the iris or ciliary processes set up by lens cortex or capsule.

Brailey (38), even while asserting that the apposition of iris and cornea is due to mechanical pressure from behind the level of the lens, observed that the latter is not necessary for the production of glaucoma. Priestley Smith (40) reported a case due, he thought, to the formation of an impervious membrane between the vitreous and aqueous chambers by the adhesion of an inflamed and thickened iris to the lens capsule, with secondary closure of the angle of the anterior chamber from pressure from behind. Stoelting (41) published a case of a woman aged 64, in whom glaucoma supervened on extraction after 14 days. The eye had to be removed three and a half months later in spite of two sclerotomies, one iridectomy and one tearing of the capsule. The capsule and the iris were found to be incarcerated in the wound, and the angle was closed all round by adhesions between iris and cornea. Stoelting ascribed the *plus* tension to traumatic choroiditis producing an increase of fluid in the vitreous which closed the angle. Treacher Collins (42) described four cases of high tension after extraction, ending in blindness and not caused by iritis, In two of these cases he found adhesion of the lens capsule and of the iris to the corneal cicatrix, and he explained the glaucoma as being the result of a forward traction on the iris, bringing it into contact with the cornea so as to block the filtration angle. Mittermaier (43) reviewed sixteen already published cases, and added to them five new ones from Prof. Becker's clinic. The glaucoma sometimes was, and sometimes was not, preceded by inflammatory symptoms. Natanson (44) collected 28 cases, and added 9 others from his own observation or from the register in St. Petersburg. He divided them into two groups: (I) Twenty-six cases. In these glaucoma appeared during the first few weeks or months of after-treatment, some having been discharged in a satisfactory condition. Entanglement of the iris or capsule was always found. (II) Eight cases. In these the glaucoma appeared several years after the extraction. Natanson quotes Becker as having examined with the microscope 38 eyes from which the lenses had been removed. In only one-third of these was the iris free from adhesions to the scar, although 32 of the eyes were removed *post-mortem*, and with no history of trouble, and Becker considered that minute adhesions may be quite undiscoverable in the living eye, and suggested the probability that such complications may

originate the glaucoma even in eyes which appear to be free from them, while he also believed that glaucoma might be primary in aphakial eyes. Natanson concluded that when the lens has been removed glaucoma is usually secondary to hæmorrhage, iritis, prolapse or adhesion of the iris or capsule in the wound, or swelling of the remnants of the lens cortex, and is usually inflammatory. It may follow any method of extraction, but probably a good iridectomy lessens the probability of glaucoma, as well as its severity, which is an argument in favor of the combined method. Knies (45) thinks that the reason for the appearance of these cases is that the iris has been insufficiently removed at the

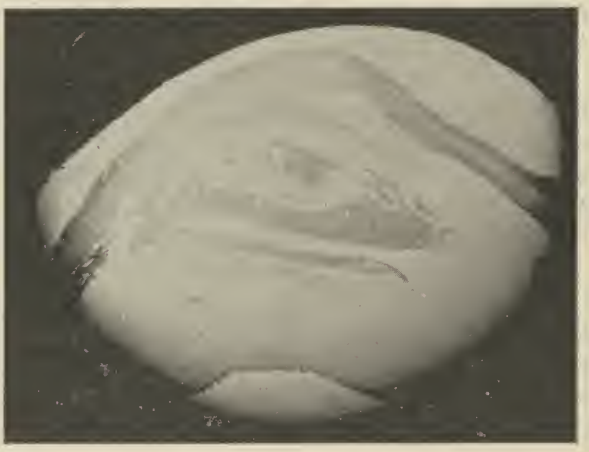

Fig. 8. Glaucoma Secondary to Extraction of Lens, and due to adhesion of the capsule to the corneal wound, a second such adhesion following on a needling, with swelling of soft remains of lens and congestion of ciliary processes.

time of the extraction. Treacher Collins (46) reported 10 more cases, and in them the prominent feature was an adhesion between the capsule and the corneal cicatrix. Schweigger (47) said "glaucoma in aphakia also witnesses against the retention theory." Priestley Smith (48) reported two cases occurring in children after the lens had been absorbed after needle operations. In one a peripheral posterior synechia united the iris to the capsule or hyaloid; in the other the pupillary margin was adherent throughout to the remains of the lens periphery, the iris, as in the other case, bulged forward, the pupil was not blocked by any visible false membrane, but the

vitreous body appeared to have opposed the free escape of the fluid from behind the iris, which also adhered to the cornea in the places where the needle punctures had been made. He also reported a few other cases in adults, due to the formation of an inflammatory diaphragm separating the posterior from the anterior chamber. Knapp (49) wrote: "Ten cases in six years, or somewhat more than 1 per cent., shows that this is not a negligible quantity." He extracted by the simple method and opened the capsule above, under the iris, and in nearly every case did a secondary needling. The iris was not incarcerated, and there was no abnormality. Treacher Collins (50) has examined since 1890 eleven more cases. At the London Hospital, Moorfields, between 1885 and 1889, .64 per cent. of eyes operated upon for senile glaucoma had been lost from increased tension. There the extractions were generally done by the combined method. Many cases are lost from serous cyclitis (or iritis), but those with which Collins was dealing were the cases which heal up and then become glaucomatous at a time varying from the union of the wounded tissues to a year or more. "In all eyes in which glaucoma comes on after extraction of the cataract there is adhesion or entanglement of the lens capsule in the extraction scar. This adhesion or entanglement holds the root of the iris, or the anterior of the ciliary processes, if the iris have been removed up to the periphery, in close contact with the back of the cornea in the region of the coloboma, and so keeps the angle of the chamber blocked in that situation. The advanced position which the capsule takes, by reason of its attachment to the cornea, draws forward the iris lying in front of it, and in this way approximates the root of the iris elsewhere than in the region of the coloboma, to the periphery of the cornea." Should the adhering tag be of inflammatory material, as sometimes happens, it is by its slow contraction that the angle becomes gradually closed. A needle operation may cause adhesion between capsule and cornea, and "leads sometimes to the onset of increased tension without any second adhesion, probably either by the swelling of the cortical lens matter so often left in the periphery of the capsule through the action of the aqueous humor on it, or by the swelling of the ciliary processes occasioned by dragging on them by tearing through the capsule. This swelling of the cortical lens matter, or swelling of the ciliary processes, may be just

sufficient to push forward the root of the iris into contact with the cornea, which is already abnormally close by reason of the advanced position of the lens capsule. I have one specimen of glaucoma occurring in an eye from which a traumatic cataract had been extracted in its capsule. In it the anterior hyaloid of the vitreous is clearly shown adherent to the cicatrix in the cornea. An adhesion of the hyaloid of the vitreous to the cornea would tend to cause a greater advance in the position of the iris than an adhesion of the lens capsule, because normally it is further back. I have met with two eyes in which the whole iris had escaped through a wound near the center of the cornea, and which afterward became glaucomatous. Until I examined sections of them microscopically the cause of the increased tension in them seemed to be quite inexplicable. In both these eyes the lens had been wounded, some portion of it escaping, a broad adhesion of its capsule to the corneal scar remaining. In the escape of the iris through the wound the anterior part of the ciliary body had become drawn forward, the advance in the position of which had been maintained by the adhesion of the lens capsule to the wound. In both specimens I find the most anterior of the ciliary processes in contact with the posterior surface of the ligamentum pectinatum."

REFERENCES CONTAINED IN CHAPTER IX.

1. Schweigger—Arch. Ophth. N. Y., XX, p. 475.
2. Brailey—R. L. O. H. Rep., IX, p. 199.
3. Brailey—R. L. O. H. Rep., X, 1880.
4. Treacher Collins—R. L. O. H. Rep., XIII, 1893, p. 502.
5. Schiess—Arch. f. Ophth., XXX, 3, 1884, p. 195.
6. Steffan—Klin. Monatsbl., 1867, p. 209.
7. Mueller—Vierteljahr f. d. prakt. Heilk., XIV, p. 57.
8. Duerr and Schlegtenda—v. Graefe's Arch., XXXV, 2, p. 83, and Ophth. Rev., 1883, VIII, p. 339.
9. Raab—Klin. Monatsbl., 1876.
10. Mauthner—"Glaucom Theorien," 1882, p. 252.
11. Pfluger—Jahresb. d. Univers. Augenh., 1882, p. 58.
12. Dufour—Horner's Festgaben, 1881.
13. Panas—"Diseases of the Eye," I, p. 522.
14. Randolph—Amer. Jour. Med. Sci., 1888.
15. Treacher Collins—Loc. Cit. (4.)
16. Warlomont—Soc. fran. d'Ophth., 1894, and Arch. Ophth. N. Y., January, 1895, p. 127.
17. Treacher Collins—Loc. Cit. (4.)
18. Treacher Collins—Lancet, Dec., 1894, p. 1464.
19. Beck—Ammon's Zeitschrift, I, 1.
20. Samelsohn—Centralbl. f. Augenh., 1880, p. 215.
21. Makroki—Arch. f. Augenh., XIV, p. 83.
22. Zinn—Klin. Monats. f. Augenh., XXVIII, p. 290.
23. Nettleship—R. L. O. H. Rep., XII, 1889, p. 72.
24. Spencer Watson—Trans. Ophth. Soc. of Gt. Brit., 1890, p. 136.
25. Brailey—Trans. Ophth. Soc. of Gt. Brit., 1890, p. 139.
26. Priestley Smith—"Glaucoma," p. 90.
27. Story—Ophth. Rev., XII, 1893, p. 6.
28. Windsor—"Diseases of the Eye," by Haynes Walton, 1875, p. 1170.
29. H. Pagenstecher—Klinik. f. Augenh., IX, 1871, p. 425.
30. Armaigiac—Rev. Clin. d'Ocul., I, 1880, p. 49.
31. Brailey—Trans. Ophth. Soc. of Gt. Brit., X, p. 139.
32. Lang—Trans. Ophth. Soc. of Gt. Brit.. 1890, p. 106.
33. DeSchweinitz—Trans. Am. Ophth. Soc., 1891.
34. Rindfleisch—v. Graefe's Arch., 1891.
35. Treacher Collins—Ophth. Rev., X, p. 101, and Lancet, Dec. 22, 1894.
36. Bowman—R. L. O. H. Rep., VI, 1865, p. 365.
37. Von Graefe—Cent. f. d. Wissensch, 1869, and R. L. O. H. Rep., VIII.
38. Brailey—R. L. O. H. Rep., IX, 1877, p. 199.
39. Priestley Smith—"Glaucoma, Its Causes," etc., 1879, p. 178.
40. Stoelting—Arch. for Ophth., XXXIII, 2, 1887, p. 177.
41. Treacher Collins—R. L. O. H. Rep., XII, 1, 1888.
42. Mittermaier—Dissertation, 1889, Heidelberg.
43. Natanson—"Ueber Glaukom in Aphakischen Augen," Dorpat, 1889, Ophth. Rev., IX, p. 140.
44. Knies—Centralbl. f. All. Path., April, 1890.
45. Treacher Collins—Trans. Ophth. Soc. of Gt. Brit., X, 1890, p. 108.
46. Schweigger—Arch. Ophth. N. Y., XX, p. 475.
47. Priestley Smith—"Glaucoma," 1891, p. 57.
48. Knapp—Arch. Ophth. N. Y., XXI, 1892, p. 293.
49. Treacher Collins—Lancet, XXV, 2, p. 1465.

CHAPTER X.

HÆMORRHAGIC GLAUCOMA; GLAUCOMA SECONDARY TO INTRA-OCULAR TUMORS; ASSOCIATED WITH DETACHED RETINA; FROM DISLOCATION OF THE LENS; FROM SWELLING OF WOUNDED LENS; SECONDARY TO POSTERIOR SYNECHIA, AND TO CORNEAL PERFORATION; AND SEROUS IRITIS.

Hæmorrhagic Glaucoma,

which must be distinguished from hæmorrhage into an already glaucomatous eye, was observed by von Graefe in 22 cases during eight years; Laqueur saw it in 3 per cent. of 268 cases of glaucoma; Risley computed its frequency at 4 affected eyes in 20,000 ophthalmic cases, while Bourgon says it occurred once in 6,741 patients at the Quinze-Vingts Hospital, Paris. It is characterized by an attack resembling that of ordinary acute or subacute glaucoma, taking place at an interval of weeks or even of many months after an intra-ocular hæmorrhage which is usually dependent upon the degenerated condition of the vascular system, due possibly merely to senility, or it may be in association with albuminuria. It may occur, however, in an apparently healthy person at an early age, and then may result from thrombosis of the central vein of the retina, as in a case of a man aged 26, recorded by Weinbaum (1). Opinions are fairly unanimious concerning its pathological anatomy. The filtration angle has usually (Knies (2), Priestley Smith (8), Treacher Collins (4), Wagenmann (5), with propulsion of lens in his three cases; Valude and Dubief (6); Weinbaum (7), etc.,) been found to be closed, but sometimes open (Valude and Dubief, 2 cases in 4, etc.), and the optic disc appears to cup in this just as readily as in other forms of glaucoma, (Valude and Dubief in 2 out of 4 cases, along with thickening of nerve sheaths; Weinbaum's case; Treacher Collins, in one case in a glaucoma of $2\frac{1}{2}$ months' duration, etc.) although the reverse has been asserted. Besides the usual anatomical characteristics of acute glaucoma, there are always present pathological changes in some of the intra-ocular vessels. In the choroid they have

been found to be sclerosed and ruptured (*e. g.*, Hache (8); Knies (9), etc.), or enormously distended with blood and with hyaline walls (Randolph (10), etc.); in one of the 4 cases published by Valude and Dubief, normal; and in the other 3, greatly distended with leucocytes. In the iris the vessels have been found hyaline (Valude and Dubief), hyaline and ruptured (Randolph), sclerosed and ruptured (Hache), and with the sheaths infiltrated, while rupture set up inflammation in the tissue of the iris, the ciliary body being similarly attacked (Wagenmann). The ciliary body may also contain enormously dilated vessels (Valude and Dubief). But the retina is the tissue whose vessels constantly show pathological changes, *e. g.*, sclerosis with closure of the lumen of even the largest, and rupture of walls (Hache, Pagenstecher), hyaline degeneration with some obliteration (Valude and Dubief), or sclerosis especially surrounding the central artery of the retina (Brailey and Edmunds (11)). And it is this retinal hæmorrhage preceding the increase of tension which has given the name to this form of glaucoma. I am not aware of any statistics which show in what proportion of cases of retinal bleeding glaucoma appears, but it seems to be the general and well founded impression that it is more frequent among them than among apparently healthy eyes. Considerable dubiety has been expressed concerning the rationale of the appearance of the glaucoma, and some unknown agent is often suspected. It must be observed that we are not speaking of hæmorrhages into the vitreous which act after the manner of a tumor by marked trespass on the vitreous chamber. But, as we have seen, most cases have all the appearances of ordinary acute glaucoma, and the most probable excessive excretion into the vitreous, combined with the congested and even inflamed state so often found in the uveal tract, seems to offer an explanation, as complete as that for any other form of the disease, of the manner in which initial retention at the angle is set up. Nettleship (12) believed that in these cases the eye was probably predisposed to glaucoma; and Treacher Collins (13) said, "the probability is that there is some unknown factor which causes both the increased tension and the hæmorrhages, and that the cases are really analagous to those of primary glaucoma." Valude and Dubief

thought that "it is probable that increase in tension of eyes affected by hæmorrhagic glaucoma depends essentially on mechanical obstruction sustained by the whole of the ocular circulatory apparatus," and that "Weber's theory, not applicable to ordinary glaucoma, is so for hæmorrhagic." For such cases as have the angle open some other explanation must be found, resembling possibly that for the high tension of serous iritis, some foundation for which may possibly lie in Case II. reported by Valude and Dubief, in which the anterior chamber was filled with exudates, and in some cases in which the iris and ciliary body have been found abnormal.

Secondary to Intra-Ocular Tumors.

Secondary glaucoma frequently sets in at a certain but indefinite stage in cases of intra-ocular tumor; but the eye may be excised without this having happened. Brailey (14) asserted that the increased tension is preceded by a very slightly diminished tension, and followed, when the eye is not removed, by a very considerable softening. He found (15) on examining 13 cases of sarcoma, 10 of glioma, and 5 of other tumors, that each one with increase tension—all but 5—had also application of the iris to the cornea; generally abnormal calibre, lessened or increased, of the *circulus iridis major;* inflammation or atrophy of the ciliary body and processes, and of the iris, as well as of the optic nerve which had not yet cupped. He had previously found (16) in a group of 10 cases a a similar condition of things. The sequence of events, according to Brailey's opinion, was that a tumor had caused a local inflammation which had spread along the vascular and fibrous tissues affecting the ciliary body, which, along with the resulting hypersecretion of fluid, pressed the iris against the cornea and closed the filtration angle. This is not quite the explanation given by Priestley Smith (17), and supported by Treacher Collins (18), who, however, agree with Brailey in finding that whenever in these cases increased tension appears the iris is applied to the periphery of the cornea, which is not found before that time. They believe that the tumor, which includes hæmorrhage between choroid and retina, obstructs the circulation probably by compression of choroidal veins (Fuchs 19), producing exudation of serum which detaches the retina. This and the advancing

growth of the tumor gradually removes the fluid part of the vitreous, leaving only the more fibrous portions, which transmit the increasing pressure from behind to the lens, zonule, ciliary body and iris, resulting in the usual shallow anterior chamber and high tension. Knies, who long ago advocated the hypothesis that not only glaucoma due to intra-ocular tumors, luxation of the lens, etc., but also primary cases, were caused by the secretion of an irritant fluid in the eye, recently renews this theory and refers to Nettleship's description of a condition resembling that named by Knies "glaucoma imminens," a cellular infiltration in the vicinity of the sinus of the anterior chamber and the optic nerve, which Nettleship observed in eyes with sarcoma of the choroid before the development of the glaucomatous symptoms. Knies found inflammation in this situation after the introduction into the vitreous of a strongly irritating fluid, in which he appears to find confirmation of his theory.

Devereux Marshall (20 a) has microscopically examined, with special reference to tension, 100 eyes enucleated for intra-ocular tumor, and concludes that the size of the growth has directly little to do with the degree of tension, increase in which is determined by diminution in size of the angle of the anterior chamber, which is most frequently closed when the choroid only is the seat of the tumor. When the ciliary body is affected diminished tension is not uncommon, and in a majority of these cases there is no increase. In most cases of glioma the tension is normal, in a few it is diminished, and in some it is raised.

Glaucoma Associated with Detached Retina.

In those rare cases in which glaucoma is associated with detachment of the retina in the absence of intra-ocular growth or hæmorrhage the increase of tension appears to be due either to iritis, secondary to choroiditis preceding the detachment, extending forward and resulting in posterior synechia, or to the formation of a highly albuminous fluid in an inflamed ciliary body, a fluid which cannot filter from the angle, and associated with a deep anterior chamber. (E. Nordenson (21), and Priestley Smith (22.)

Glaucoma from Dislocation of the Lens.

The lens when displaced *into the pupil* is apt to cause increase of tension by interfering with the forward passage of fluid and when *into the anterior chamber*, the iris

being pressed forward against it and against the cornea, a similar difficulty results at the pupil and also at the angle, which may at once disappear on the lens resuming its normal position when the patient lies on his back, or when the pupil is sufficiently dilated by atropine. When the lens retains its positlon behind the iris, but is dislocated *to one side*, it has been found to cause

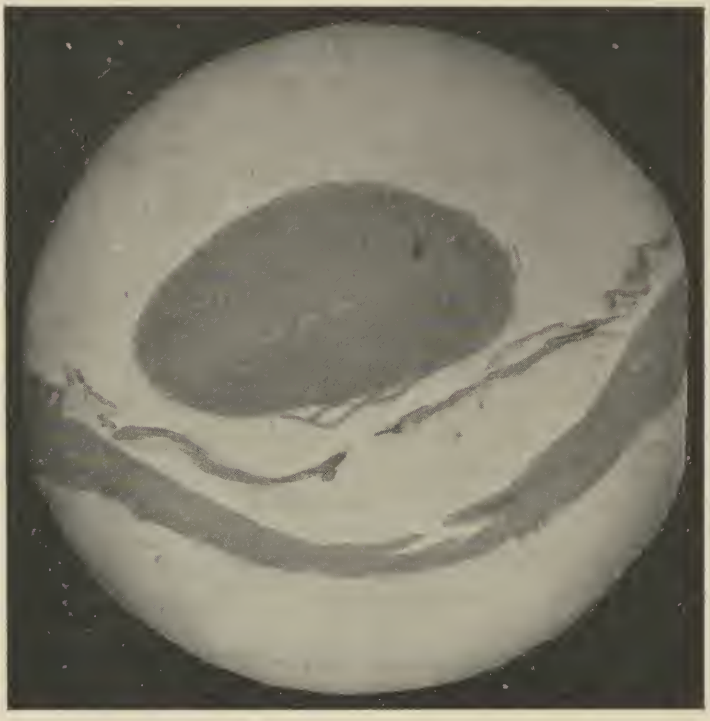

Fig. 9. Glaucoma Secondary to Lateral Dislocation of Lens. The suspensory ligament was torn, and the vitreous protruded between the lens and the ciliary body.

glaucoma by being forced by the vitreous against the ciliary body and iris, whereby these are brought in contact with the cornea in a greater or less segment of the circumference, or even the whole of it through the additional agency of the vitreous itself at the place vacated by the lens (Priestley Smith, 23). When displaced *backward* the *plus* tension is due to a "relative alteration of position which has taken place between the lens and the vitreous body directly forcing forward the root of the iris into

contact with the cornea, so closing the angle of the anterior chamber, for in these cases the angle is always found closed." (Treacher Collins, 24.) Galezowski (25) in two cases of dislocation of the lens downward concluded that the resulting glaucoma was due to obstruction by the lens of the lymph channels. The above is

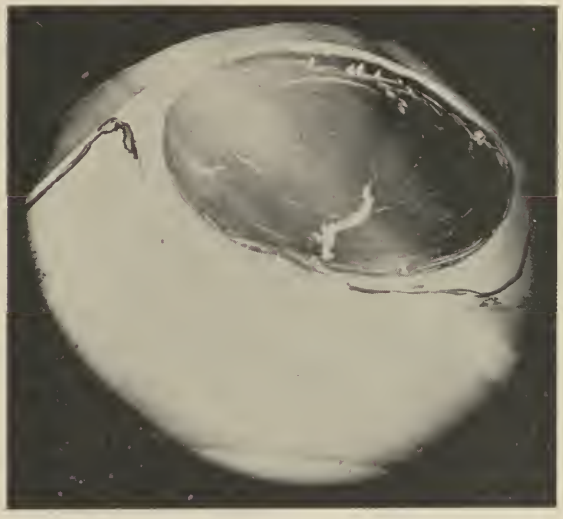

Fig. 10. Glaucoma Secondary to Dislocation of Lens Into Anterior Chamber. Angle on one side blocked. Eserine in this case increased the tension, and atropine diminished it.

undoubtedly the best founded explanation of these cases, though Schweigger (26) opposed it and asserted, on what scientific grounds I do not know, that the increased tension is due to an irido-choroiditis; and Brailey (27) suggested long ago an iritis "of the same kind as iritis serosa."

Swelling of Wounded Lens.

After an acccidental or intentional wound of the lens the eye has to be carefully watched on account of the possibility of increased tension, due to blocking of the pupil by lens matter or displacement of the iris forward. This is not at all an uncommon occurrence after for needling lamellar cataract. The removal of the lens will permit of a return of the normal tension.

Glaucoma Secondary to Posterior Synechia.

There is little doubt that the usual explanation of the sequence of glaucoma upon total exclusion of the pupil, by

adhesion of the iris to the anterior capsule of the lens, is the correct one, viz., that in this way fluid is prevented from leaving the posterior to enter the anterior chamber, the iris is bulged forward, "bombé," and when left to itself becomes adherent at its periphery to the cornea, while the high tension causes atrophy of the ciliary body, etc.

It is true that it sometimes happens that apparent total posterior synechia exists without *plus* tension, but it must be borne in mind in these cases either that the completeness of the synechia may be only apparent, or that diminished excretion from the ciliary body due to atrophy after participation in the inflammation which caused the synechia, or atrophy of the iris itself permitting of the passage through it of fluid, will generally account for this anomaly. Against the above theory of this form of secondary glaucoma a few dissentient voices have been raised. For instance, Schweigger (28) and Rheindorf (29) asserted that a dragging on the ciliary body would account for all the symptoms by causing hyper-secretion of fluid; and Ulrich (30), who held that normally fluid passes forward into the anterior chamber through the iris, considers the usual explanation much too simple. According to him in the iris thickening of tissue and destruction of vessels take place, making it impervious, and also causing a collateral hyperæmia with hyper-secretion from the ciliary processes, the latter being more important than the retention. That there should be any fiuid at all in the anterior chamber in these circumstances proved to his mind that it passed through the iris.

Glaucoma Secondary to Corneal Perforation.

The adhesion of the iris, lens capsule, or vitreous to the cornea as a result of ulceration or wound of the latter is apt to result in glaucoma after the closure of the perforation has stopped leakage from the eye. The same thing may be seen in staphyloma of the cornea or ciliary region, the bulging perhaps increasing for a time till the wall gives way, and lowered tension permits of healing when the same process is repeated, possibly until by the access of infective material to the interior of the eye, a uveitis is set up which ends in complete destruction. Brailey (31), who reported on the examination of 50 cases of glaucoma following on perforation of the cornea, was of opinion that

increased tension was not directly due to the universal presence of a closed angle, but that the hypersecretion with which it was associated from dragging on the ciliary body was primarily of consequence. Ulrich's (32) idea as to the etiology of such cases, published at different times, differs from Brailey's. He founded his theory mainly upon observations on a case of gonorrhœal ophthalmia without necrosis of the cornea and complete posterior synechia, in which after five months the eye had healed over with good perception of light and normal tension; and of eleven cases of artificial removal of the cornea with adhesion of the iris to the scar in rabbits. Four of these eyes became glaucomatous at times varying from one to three months after the production of leucoma, with bulging of the cicatrix, etc. In five cases the increase was inconstant or uncertain, and in the remaining two the globes softened. The differences noted in the condition of the glaucomatous eyes from that in the others were that in the latter the pupillary portion of the iris was less adherent, and that there was less deposit of pigment in the ciliary processes, showing less chronic hyperæmia. Still hypersecretion had been demonstrated during life in both sets of cases, but though Ulrich claims for it the important part in developing the glaucoma it did not do so in the majority of these. In the gonorrhœal cases *plus* tension came on as soon as fibrous degeneration of the exposed iris had developed. He explained his theory in these words (33): "The immediate effect of an extensive incarceration of the iris in the corneal defect is, besides the mechanical imprisonment, traction upon the iris and the corresponding part of the ciliary body, and partial narrowing of the anterior chamber and Fontana's spaces. We notice an immediate hyperæmia of the ciliary processes and an increased production of the aqueous humor. The mechanical incarceration of iris tissue acts directly, and the secondary degeneration and atrophy act indirectly to produce disturbances of the circulation in the iris. My experiments on animals have convinced me that incarceration of the pupillary portion of the iris produces glaucoma more readily than peripheric incarcerations. The reason for this seems to be that the former encloses a larger vascular area than the latter." In considering these observations of Ulrich's one comes to the conclusion that, however

interesting they may be, they fail adequately to support his theory, if he means by it that hypersecretion without retention suffices for the production of the increase of tension, because he admits hyper-secretion in both the glaucomatous and the non-glaucomatous groups; because, as is well known, atrophic irides, such as these were, may easily allow of the filtration of fluid to the angle of the anterior chamber; because normal or sub-normal tension may result in such cases from the degeneration of the ciliary body which is so likely to accompany a similar pro-

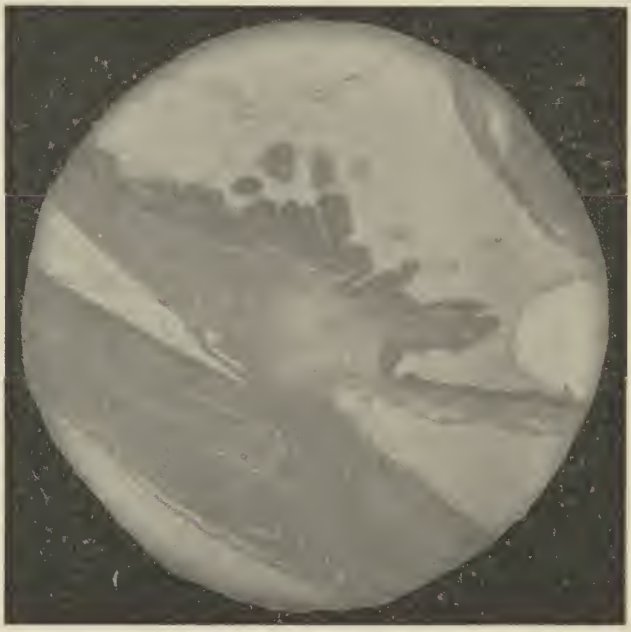

Fig. 11. Sympathetic Cyclitis with High Tension and Deep Anterior Chamber, due to excessive secretion and blockade of the angle of the anterior chamber by cellular matter. The Lens was cataractous.

cess in the iris; and because minute fistulæ in the cornea, easily overlooked, might admit of a constant drainage of fluid. Priestley Smith (34) gave illustrations, among his other excellent drawings, of the best explanation of glaucoma secondary to corneal perforation, viz., that on the emptying of the anterior chamber iris, lens, etc., are pressed forward, and the iris, or even the lens capsule, becomes entangled and ultimately adherent in the corneal wound, which if tolerably complete, will prevent the

fluid from passing round into the anterior chamber, or simply by bringing the periphery of the iris in contact with the cornea, when less complete, block up the filtration angle.

Serous Iritis.

In serous iritis it is now understood that the ciliary body is principally at fault. Into the symptoms of this disease it is unnecessary to go here, beyond remarking that it is frequently characterised by increased intra-ocular tension along with an anterior chamber as deep as, or deeper than, normal. On section there is always found much cell infiltration near Schlemm's canal and in the ciliary body (Brailey (35)), without atrophy of the ciliary muscle (Brailey and Edmunds (36)). While serous iritis is not included among the forms of primary glaucoma it may yet lead to a secondary form of the disease with deep cupping. The cause of the increase of tension is believed to be a change from the normal in the composition of the aqueous fluid, whereby it contains, besides pigment and leucocytes, an excess of albuminous matter which result in a diminished power of filtratiou into the channels of exit, especially when these are more or less blocked by cells (PriestleySmith 37, Treacher Collins 38). This is a true example of a *plus* tension due primarily to a block at the angle of the anterior chamber, and may be compared in its effects on the depth of that chamber with the *plus* tension due to a hindrance in a similar position according to the theory of Knies.

REFERENCES CONTAINED IN CHAPTER X.

1. Weinbaum—Arch. f. Oph., XXXVIII, 1892, p. 191.
2. Knies—Arch. f. Oph. XXII, p. 163.
3. Priestley Smith—"Glaucoma," 1891, p. 128.
4. Treacher Collins—R. L. O. H. Rep., XIII, 1893, p. 500.
5. Wagenbaum—Arch. f. Oph., XXXVIII, 1892, p. 213.
6. Valude and Dubief—Ann. d'Ocul., Aug., 1892.
7. Weinbaum—Loc. Cit. (1).
8. Hache—Recueil d'Oph., 1875.
9. Knies—Centralbl. f. All. Path., April 1, 1890.
10. Randolph—Trans. Am. Med. Ass., 1891, p. 410.
11. Brailey and Edmunds—R. L. O. H. Rep., X, p. 86.
12. Nettleship—R. L. O. H. Rep., XII, 1889.
13. Treacher Collins—R. L. O. H. Rep., XIII, 4, p. 494.
14. Brailey—R. L. O. H. Rep., X, 2, p. 275.
15. Brailey—R. L. O. H. Rep., X, 1877, p. 225.
16. Brailey—R. L. O. H. Rep., IX, p. 199.
17. Priestley Smith—"Glaucoma," p. 64.
18. Treacher Collins—R. L. O. H. Rep., XIII, 4, 1893, p. 493.
19. Fuchs—"Das Sarcom des Uveal Tractus," Vienna, 1882, p. 210.
20. Knies—Arch. Oph., 1895, XXIV, 2, p. 184.
20a. C. Devereux Marshall—Trans. Brit. Oph. Soc., XVI, 1896, p. 155.
21. Nordenson—"Die Netzhaut Abloesung," Wiesbaden, 1877.
22. Priestley Smith—"Glaucoma," p. 71.
23. Ditto, p. 55.
24. Treacher Collins—R. L. O. H. Rep., XIII, p. 491.
25. Galezowski—Bull. de l'Acad. de Med. de Paris, XXXI, 1894, p. 491, Rep. by Chauvel.
26. Schweigger—Arch. Oph. N. Y., 1891, XX, p. 475.
27. Brailey—R. L. O. H. Rep., IX, 1877, p. 199,
28. Schweigger—Loc. Cit. (26).
29. Rheindorf—K. M. f. Aug., XXV, 1891.
30. Ulrich—Arch. f. Augenh.. XXVI, 1892, 3, p. 1.
31. Brailey—Loc. Cit. (27).
32. Ulrich—V. Graefe's Arch. XXX, 4 and Loc. Cit. (30) and Arch. Oph. N. Y., 1893, XXXII, p. 475, and Oph. Rev. XIII, 1894, p. 135.
33. Ulrich—Oph. Rev., XIII, p. 137,
34. Priestley Smith—"Glaucoma," p. 47.
35. Brailey—R. L. O. H. Rep.. X, 1880, p. 94.
36. Brailey and Edmunds—R. L. O. H. Rep., X, 1880, p. 85.
37. Priestley Smith—"Glaucoma," p. 36.
38. Treacher Collins—R. L. O. H. Rep., XIII, p. 493.

CHAPTER XI.

SIMPLE GLAUCOMA.

Simple glaucoma was said by von Graefe (1) to affect almost without exception both eyes successively; Nettleship (2) stated that in something like two-thirds of their number cases of chronic glaucoma are sooner or later bilateral; Stedman Bull (3) thought that probably in 80 per cent. both eyes become affected. We have seen that the symptoms of glaucoma may pass by slow gradations from those of an excessively acute disease, marked by great pain and every appearance of congestion, and leading rapidly to loss of vision, to a condition in which all such signs are lacking, and little or nothing is apparent to denote a deviation from the normal beyond a certain visual deficiency associated with cupping and pallor of the optic disc, in many cases a picture hard to be distinguished from that of optic atrophy. These latter facts have given to simple or chronic glaucoma a peculiar interest, which, though it existed in full force in von Graefe's time, has diminished little to the present day in spite of the great advances which have been made in recent years in our knowledge of glaucoma as a whole. This interest centers chiefly round three points, viz.: (1) Is this disease really glaucoma at all? or is it something else? (2) If it is a glaucoma, will explanations applicable to the acuter forms of the disease meet the case of this also? or must some other be produced? And (3) is it advisable to apply the same treatment for this as for the more distinct types?

The first two of these questions at once lead us to the observation that there has long been, and still is, a divergence of opinion on the subject among the most eminent ophthalmologists. Thus, for example, von Graefe said he could not too strongly insist "that in the present state of our knowledge the optic nerve affection alone is not sufficient to warrant a diagnosis of glaucoma; in many instances it has a patho-genesis entirely distinct from the latter disease." He called it "amaurosis with optic nerve cupping," while Edward Jaeger and Stellwag marked their uncertainty by designating it respectively "glaucomatous optic nerve disease" and "excavation atrophy."

Jacobson (5) separated glaucoma simplex from all other forms, as did Knies (6), who said that von Graefe's designation was still applicable, both clinically and pathologically. Nettleship (7), while referring to the "disc appearances of glaucoma where there is none," and to the possibility of atrophic changes in a nerve head in which there had previously been a large physiological cup, producing an excavation resembling that of glaucoma, yet bore strong testimony to the unwisdom of the least carelessness in diagnosing as some other disease cases having discs resembling those of glaucoma, simply because the disc appearances may be the only ones suggestive of glaucoma, which the cases may ultimately prove themselves to be by the development of other and distinctive features; or, in his own words, "we cannot but be very careful in inferring that a person whose discs show changes resembling those of glaucoma is really safe, however free he may be from symptoms or other signs of the disease." Nettleship illustrated by a few examples the wide range in the duration of chronic glaucoma before the accession of blindness, which, however, occurs usually within two years, thus:

Captain ———, myopic 2.5 D, had after simple glaucoma for 20 years a visual acuity of $^{6}/_{9}$; he then was acutely attacked, and was blind three years later. Miss ——— was glaucomatous for 9 years before her vision was very materially affected. Mr. R. was blind in the right eye from glaucoma, and, while at the age of 76 the left had been subject to the disease for 6 years with T +, vision was still $^{6}/_{18}$. As suspicious cases, which had not developed at the time of publishing his paper, the author gave: Mrs. MacR., age 48. In 1881 both discs were very suspiciously cupped, and in 1885 had not changed, the vision also remaining $^{6}/_{6}$, H. M.—0.75 D. and T.n; also Mrs. B., age 57, in 1884, M. vision with glasses $^{6}/_{18}$, discs cupped and pale, T + slightly; in 1888 sight was still good. "That such suspicious appearances may precede the outbreak of rapid glaucoma is proved by" Mrs. G., age 40. In September, 1885, right eye had central, large, very steep "physiological" cup, quite different from that in the left. The right eye suffered from subacute glaucoma three months later. Cases also occur of real glaucoma in one eye, with very deep and large normal cups in the other, as that of Dr. ———, one of whose eyes was excised for absolute simple glaucoma, with retinal hæmorrhages and pain. For the previous two and a half years he had had a pale disc, cupped all over to the depth of 1 m.m. Vision $^{6}/_{6}$; field full, T.n. There were at one time hæmorrhages in this eye, also, which absorbed. In Edward S's case an iridectomy in 1883 cured subacute glaucoma in the right eye. Two years later the disc presented a large physiological cup, which was rather pale. The left, with vision of $^{6}/_{6}$, had a deep physiological cup. Mr. G.,

age 50, had simple glaucoma of the left eye, with contracted field. Right had perfect field and no symptoms; discs much alike, having large cups on the external sides and broad belts of nerve tissue on the internal. John S., age 51, in August, 1879, had advanced chronic glaucoma in the left eye, while in the right there was no glaucoma. Vision $^{6}/_{9}$, with —.75 D, a large, circular and very deep physiological cup. Three years later the left eye was quite blind and the right remained as before. These cases show that even "physiological" cups must at times be viewed with suspicion of glaucoma.

Nettleship suggested that natural variations of resistance in the lamina cribrosa may have effect in producing abnormal disc appearances, and said: "I think it likely that a very large physiological cup shows a predisposition to the further yielding that will constitute glaucoma." Then one sometimes sees cases of simple glaucoma in which the "first thing observed is a nearly central defect in the form of one or more scotomata near to or partly surrounding the center, and without any contraction of the periphery. These cases are amongst those as to the nature of which there is much difference of opinion, many surgeons looking upon them as peculiar varieties of primary uncomplicated atrophy of the optic nerve rather than glaucoma." Nettleship, while admitting that this is a "difficult point to settle," quoted a case to show that "although in some of the cases of very quiet, doubtful glaucoma T is never decidedly raised, and variations of sight are absent," an "early failure of the central part of the field is compatible with both these cardinal symptoms of glaucoma."

Priestley Smith (8) said: "Some writers, however, still deny that what we call glaucoma is necessarily connected with increased pressure. They point to certain exceptional cases in which the contracted field and the excavated disc are found in company with normal tension, and on the strength of these exceptions they maintain that glaucoma is the expression of some unknown agent which usually raises the pressure and excavates the disc at the same time, but which occasionally excavates the disc without raising the pressure. This assumption is, I think, unnecessary. The excess of pressure is sometimes slight; it is often intermittent; it may even be absent for long periods of time; it is very probable, therefore, that these cases of glaucoma with normal tension are cases which have been examined only during the intermissions of in-

creased tension. Permit me to mention a case: Sir William Bowman was consulted by a lady in 1865. He noted—and he has kindly placed the notes at my disposal—increased tension and commencing excavation, diagnosed glaucoma, and spoke of iridectomy. No operation was performed. Twenty years later this lady came under my own care with deeply excavated discs, contracted fields, and impaired vision. I saw her many times; sometimes the tension was quite normal, sometimes it was decidedly increased," (*e. g.*, one day at 5 p.m. quite normal in both eyes, at 11 p.m., when tired, decidedly in excess, the pupils being larger than before); "vision was still useful, but failing rather rapidly." Iridectomy was performed on both eyes with good result. "Now, this lady must have had for years an unmistakable glaucoma, but at times, and probably very frequently, a normal tension. Yet, pressure was there at the beginning, and it was there at the end. I cannot doubt that it was an essential factor in the process." Now, according to Priestley Smith's own teaching, if there is pressure there should also be apposition at the periphery between iris and cornea, and yet we know that the anterior chamber has sometimes the appearance of normal depth in these cases. But, as this author remarks, eyes are not removed for simple glaucoma until a marked increase of tension and pain have supervened, or, should they be, the very excision would probably permit the parts to resume their natural positions, as it is well known sometimes happens. And also the mere fact that the shallowing of the anterior chamber is not visible from without in no way proves that the iris and cornea are not in contact where the former is cut off from view behind the corneo-scleral junction. It is sometimes seen in eyes excised for glaucoma that at the point where the adherent iris leaves the cornea it forms an abrupt bend, which would tend in the living eye to give an appearance of depth to the anterior chamber, and Treacher Collins (9), who has noted this peculiarity, observed, like Priestley Smith, that a closed angle is quite compatible with a deep anterior chamber. Stedman Bull (10) found the anterior chamber generally shallow, and the iris more or less mobile, except where the glaucoma was absolute. The tension was normal in both eyes in 2 out of 90 cases. The interval between the attack on the first and that on the second eye varied from 2 months to 12 years in the 26

cases in which this could be accurately ascertained; in 7 eyes no cup was visible. Among this large number of cases there was apparently little difficulty in diagnosing all as glaucoma, and most of them seem to have had a typical shallow anterior chamber and increased tension. Rheindorf (11) thought v. Graefe's designation of "amaurosis" still applicable to simple glaucoma, which is not glaucoma at all; also that in some of these cases the distinctly high tension is due to a continuation of an increased brain pressure into the eye in cases of syphilis, traumatism or hydrocephalus. Schweigger (12) has strongly upheld the theory that only a certain number of cases of so-called glaucoma simplex have any right to be classified as such, in which, as he says, increase of tension is a necessary feature. Glaucoma simplex should be divided into (1) true glaucoma without inflammatory signs, and (2) diseased optic nerve with pre-existing physiological excavation. "The ophthalmoscopical picture of a physioiogical cup may be produced by glaucoma, just as extensive normal excavation with discoloration of the disc cannot be distinguished from excavation due to intra-ocular pressure." "Physiologial excavations are always bilateral, but are not always identical in size and location." While Schweigger admitted the possibility of glaucomatous excavation without any sign of inflammation in the eye, he considered any abnormal condition of the pupil as an indication of a past inflammatory attack. "I conclude," he said, "that the course of the disease is marked by distinct attacks of increased tension which come on, and (in the early stages at least,) again subside," some of them apparently non-inflammatory. The writer has seen in Prof. Schweigger's clinic in Berlin several cases diagnosed as optic atrophy, which would certainly by many ophthalmologists have been named glaucoma. Schweigger's opinions may usefully be compared with what has been quoted above from Nettleship. Fuchs (13), holding that glaucoma simplex *is* glaucoma, though occasionally difficult to diagnose from optic atrophy with exceptionally deep excavation, says that in these cases where increase of tension is very doubtful, we must suppose a "lamina cribrosa of special weakness, so that it will give back before a pressure not markedly raised above the normal." We have already seen that such a hypothesis may be unnecessary, and that, although hidden from outward inspec-

tion, the changes found at the anterior part of the eye in acute glaucoma are probably usually present also in a mild degree in the simple chronic form. But the frequently apparently normal condition of the anterior section, and the comparatively constant pathological changes at the disc have led certain authors to associate true simple glaucoma entirely with a diseased condition of the optic nerve or its sheaths, where they think it begins and may end. This supposition has been already considered, and the conclusion formed that it is greatly wanting in substantial evidence. Zentmayer and Wm. Campbell Posey (14), assistant surgeons in the Wills Eye Hospital, Philadelphia, have recently published a careful analysis of 167 cases of simple glaucoma which have been observed there in the clinics of Norris and Oliver. This number represents .736 per cent. of 22,680 patients, 10,591 of whom were males and 12,089 females, with 85 male and 82 female cases of simple glaucoma. The notes made by Zentmayer and Posey bear out the already known fact that, though simple glaucoma occurs occasionally early in life (they noticed a case at 13), it is markedly more frequent after 50, and from 55 to 65 is its favorite decade. They endeavored to discover whether any systemic disease has any noticeable tendency to its production, and concluded that 21 cases of rheumatism and 7 of influenza were a sufficient basis for placing these in that category. Of the 167 cases 39 sought treatment on account of failing sight, 19 on account of presbyopia, 19 on account of ocular pain, 5 on account of headache, and 3 on account of asthenopia, and in a number of cases the disease was accidentally discovered in patients suffering from conjunctivitis and other affections. In 36 glaucomatous eyes central visual acuity was normal. In only 13 cases was there normal vision in the whole field of one of the eyes. On an average 20 months elapsed after the attack on the first eye before the second became affected, and $3\frac{1}{4}$ years between the first signs of failing sight and the time at which a surgeon was first consulted. About $2\frac{1}{2}$ years was the average interval between the first appearance of the disease and the total blindness. In 32 eyes with full visual acuity there were distinct glaucomatous excavations. The anterior scleral vessels were engorged in 28 instances, the cornea was steamy or "needle-stuck" in 58, anæsthetic in 22, irregular and maculated in

10. In 64 eyes the anterior chamber was shallowed and the disc excavated. In 10 cases the anterior chamber was shallow and the disc not excavated. In 3 eyes the disc was excavated, but the anterior chamber was normal. In 15 cases where the anterior chamber was markedly narrowed the tension was noted as being normal. "The shallowing of the anterior chamber exists in eyes where there is no excavation, but the converse is not true, for in every eye where there is an excavation the chamber will be shallowed." * * * "The larger the pupil the less will be the reaction to light." Increased tension was noted in 32.63 per cent. of the cases. In only 4.52 per cent. were there signs of irritation in the anterior segment of the eye. In 35.6 per cent. of the cases with excavation there was also enlarged pupil. Lenticular opacities existed in 95 instances, and vitreous opacities in 6. Where there were excavations the disc was always discolored, from a reddish gray, the most common, to a blue and green; and the average depth of the cup was 3.37 D. The total number of eyes possessing normal tension and pathological excavation was 72. The form field was contracted on the average to 34.83° on the nasal side, to 50.10° on the temporal, to 31.75° above, and to 43.71° below. The contraction for red was to 20.53° on the nasal side, to 31.75° on the temporal, to 19.35° above and to 24.52° below. Seventeen cases in which the form field was abolished gave an average contraction for the candle field to 26.27° on the nasal side, to 33.81° on the temporal, to 34.81° above, and to 40.45° below. In the majority of cases the contraction was concentric and proceeded *pari passu* for form and color, but in 32 the field for color was less than that for form. In 24.46 per cent. there was a tendency for the field to be cut in the form of sectors, but in no one special position. Scotomata were noted in four instances. By a comparative study of 121 cases the authors concluded that a diminution in vision, a contraction in the field, and the presence of excavation go hand-in-hand. A spontaneous venous pulse occurred in 22 cases, (a remarkably small proportion) and an arterial in 14. In about 20 per cent. of the cases the choroid was described as congested. Hypermetropic eyes were much more frequently attacked than emmetropic and myopic, but not so relatively to their frequency in the general population, and high degrees were no more liable than low degrees, 1.66 D.

being the average. "From observations elsewhere the authors are confident that astigmatism against the rule occurs in the vast majority of cases of glaucoma, and it has been a great disappointment that this belief was not substantiated by this series of cases."

We are taking it for granted that all these 167 cases were pure glaucoma, though the authors do not say in what way, *e. g.*, the 72 eyes with "pathological excavations" and normal tension were differentiated from optic atrophy, or what kind of pathological excavations existed in the 32 eyes with normal visual acuity. These points are tc be noted because the authors had previously stated generally that the excavations were of all varieties, and varying degrees of depth and extent. Sixty-five were complete, 7 were undermined, 9 shallow, involving the entire surface. They go on to draw from these cases the very important conclusion that, "the relative amount of contraction in the form and color field often adopted in the distinction between an atrophic and a glaucomatous excavation is valueless, as the findings show that in quite a large proportion 13.06 per cent. of the cases the color field was relatively more affected than the form. "Indeed in 16 of these 32 cases (50 per cent.) the form field was normal, whilst that for red was contracted to 20 or less. Contrary to the findings of other observers, the most frequent type of the restriction of the visual field consists in concentric limitation of the entire field and in the contraction to the nasal side."

Whatever be our belief concerning the etiology of simple glaucoma, the fact remains that practical surgeons occasionally meet with cases which they find difficult of diagnosis and an important question arises:—Is there any known means by which the two diseases can be definitely separated?

REFERENCES CONTAINED IN CHAPTER XI.

1. Von Graefe—R. L. O. H. Rep., XII, p. 104.
2. Nettleship—R. L. O. H. Rep., XII, p. 79.
3. Stedman Bull—Trans. Am. Oph. Soc., 1889.
4. Von Graefe—Arch. Oph., III, 2, 1857.
5. Jacobson—Arch. f. Oph., XXXIV, 1, p. 169.
6. Knies—Centralbl. f. All. Path., 1890, I, p. 249.
7. Nettleship—R. L. O. H. Rep., XII, 1888, p. 72.
8. Priestley Smith—"Glaucoma," p. 2, and Ophth. Rev., 1885, p. 261.
9. Treacher Collins—Lancet., 1894, p. 1465.
10. Stedman Bull—Loc. Cit. (3).
11. Rheindorf—Klin. Med. f. Aug., XXV, 1891.
12. Schweigger—Arch. Ophth., N. Y., 1891, p. 475.
13. Fuchs—"Lehrbuch der Aug., p. 389.
14. Zentmayer and Posey—Arch. Oph., N. Y., XXIV, 1895, p. 378.

CHAPTER XII.

DIAGNOSIS AND PROGNOSIS.

Diagnosis.

While the principal difficulty in diagnosis lies in distinguishing simple glaucoma from optic atrophy, there are a few points which have been already briefly referred to, and which are worthy of mention again in this connection, so much evil having arisen from a failure, on the part chiefly of the general practitioner to, correctly understand cases, especially of acute glaucoma.

First, it should be noted that glaucoma may be attended by symptoms of fever; that the skin in the neighborhood of the orbit may have an erysipelatous aspect; that the pain may resemble that of rheumatism or neuralgia of that side of the head, and even extend to the shoulder, and that when associated with vomiting it may provoke a diagnosis of sick headache, etc. Locally, it may be mistaken for some form of conjunctivitis, for keratitis, for iritis, for cataract, for opaque vitreous, or for optic atrophy. In all these increased tension, which is always present in cases of glaucoma which are at all acute, will at once direct the surgeon in the right direction, as will cupping of the disc or retinal pulsation when present. From conjunctivitis it can further be distinguished by the opacity of the cornea, the shallow anterior chamber, and by the dilated and comparatively motionless pupil, as well as by the enlargement of the ciliary vessels; from keratitis by an absence of constitutional cause, the position and nature of the corneal opacity, and the state of the pupil; from iritis by dilatation of the pupil, which in iritis is contracted, by the absence of posterior synechia and the depth of the anterior chamber. The possibility of "iritis serosa," properly cyclitis, with raised tension and deep anterior chamber, virtually a form of glaucoma, should be borne in mind. Glaucoma is to be distinguished from cataract because glaucoma with a clear lens does not really resemble cataract in any respect; on the other hand, glaucoma may arise from the swelling of a wounded lens, when the history and appearance of the lens should suffice for guidance. But further, glaucoma

may supervene in a cataractous eye, or cataract may supervene in a glaucomatous eye. Both of these conditions should be at once apparent to the careful examiner, though he may be in some doubt as to which had priority in point of time. "The cataracta glaucomatosa reveals itself in marked swelling, bluish-white color, and a lively silken reflex from its surface, whilst cataract in oculo-glaucomatoso possesses the appearance which corresponds to its origin and nature"; in opaque vitreous the cornea and lens are clear, while the opacities themselves are visible.

Besides the above points of difference the history must always be considered; while in certain cases other symptoms of glaucoma not mentioned, such as true glaucomatous halos, may prove of assistance in arriving at a correct conclusion. In those uncommon cases where a diagnosis cannot be made without difficulty between glaucoma and optic atrophy, owing to the absence of satisfactory symptoms in the depth of the anterior chamber, in the size and shape of the pupil, in the condition of the fundus and of the tension, one has to rely upon certain other points which, however, do not in every case with sufficient distinctness supply the desired information. Thus, visual fields are in glaucoma more usually contracted on the nasal side, the retention of peripheral color vision is longer in glaucoma, in which also the light minimum is much higher than normal, while the light difference is apt to be little affected, the opposite of which obtains in optic atrophy. When a large "physiological" cup is found in one eye only, it is really glaucomatous. Also, very slight pressure on the globe may produce pulsation in the retinal arteries in glaucoma. In the discussion on the subject of simple glaucoma which was introduced by Nettleship at the meeting of the British Medical Association in London in 1885, he put the question whether optic atrophy with "glaucomatous" cupping, occurring during the glaucomatous period of life, unaccompanied by true color blindness or by the smallest indication of disease of the central nervous system, linked also by imperceptible gradations with typical chronic glaucoma should be looked upon as glaucoma or as optic atrophy? For himself, he answered that when the disc shows well marked glaucomatous cupping the case should almost without exception be looked upon and dealt with as glaucoma, whether in-

creased tension and other common signs be present or not. Probably where the tension is low the lamina cribrosa is weak, and yields to slight pressure. Meyer (Paris) could not agree with Nettleship, and thought such cases were optic atrophy. Priestley Smith desired a greater agreement in a definition of glaucoma, and said that it meant to him a morbid process depending essentially upon raised intra-ocular pressure. He advocated the more general use of the tonometer as an aid in settling the question. In a recent address before the Academy of Medicine of Paris, Galezowski has endeavored to clearly differentiate between ataxic and glaucomatous atrophies of the optic disc. He affirms that if the affection is glaucomatous, 1, the disc is white, more especially on the external side; 2, the central veins of the optic nerve are diminished in size at the point of emergence, but after crossing with the central arteries, most frequently near the edge of the disc, they become larger; 3, pulsation of the central artery is seen; 4, the pupil contracts to light but is enlarged and irregular; 5, the visual field is narrowed in the internal or superior-internal portion, while it remains normal, at least for a long time in the external and inferior portions; 6, dyschromatopsia does not exist at the beginning of the disease, and is hardly appreciable later, when the opaque zone of the internal visual field has reached the central point of vision; 7, the patella reflexes are preserved and there are no shooting pains. The disease is of gouty, arthritic, or syphilitic origin. Repeated anterior sclerotomy and general treatment appropriate to the diathesis considerably improve the atrophy of the disc and sometimes even arrest it, as Galezowski has seen in five out of seven cases which have come under his observation.

Reasons Why the Quietest Cases of Simple Glaucoma May Be Real Glaucoma.

We may take it for granted that in doubtful cases the distinct appearance of high tension or of peripheral synechiæ would settle the question in favor of glaucoma, and also that there is no reason why glaucoma and optic atrophy should not co-exist in the same eye. It will also be admitted that it is frequently hard to say whether the tension is raised or not. Brailey thought that many cases of so-called optic atrophy were really glaucoma simplex with Tn. or T?+, and Schweigger (1) has as-

serted that unless the increase of tension is distinct this symptom is of no value, for one can easily discover high tension if one have only faith enough. It is also known that the normal ocular tension varies with the individual. It is, therefore, certain that what is a normal tension for some eyes is a *plus* tension for others, and it must be just as easy to omit to find a real *plus* tension as to imagine one which is not there. Then how is it that we encounter cases in which there co-exists a combination of a normal tension with glaucomatous halos or rainbows? (*e.g.* Somya 2, and Brailey 3), for we have seen the latter to be due to a slightly œdematous condition of the cornea produced by an unwonted pressure from within the eye. Cases also occur in which the tension is noted as normal, and yet the cornea and iris are in contact at the periphery (*e. g.* Bitzos 4) which is now generally believed to be due to pressure from behind. This combination also appears to militate against the theory of an abnormally weak lamina cribrosa. The statement made by Zentmayer and Posey (5) in describing 167 cases noted in the clinics of Norris and Oliver, that "shallowing of the anterior chamber exists in eyes where there is no excavation, but the converse is not true, for in every eye where there is an excavation the chamber will be shallowed," appears to go a long way toward proving that *plus* tension—as witnessed by the shallow anterior chamber—is or has been always present where there is a true glaucoma cup, *plus* tension meaning an increase of tension as regards the eye involved, and not an increase as measured by a supposed universal normal. It is possible that even the recumbent position at night, the tiredness from the day's work, (glaucomatous patients being generally at their best in the morning) etc., may produce temporary *plus* tension sufficient in the long run to seriously affect the eye. When we know that in the majority of cases of chronic glaucoma heightened tension is distinctly present at one time or another, and that acute glaucomatous attacks not infrequently supervene in such eyes, we should be very chary in throwing over glaucoma because we may not be able to discover any appreciable hardness of the globe. We have seen that in all probability there is an automatic action within the eye whereby a rise of intra-ocular tension due to hypersecretion has the effect of again bringing the tension to normal through pressure on the uveal vessels, preventing further secretion

from them. It would appear as if in the quietest forms of simple glaucoma the blood pressure were so low that a comparatively slight rise of intra-ocular tension would attain this result, secretion being again diminished; or, given some hindrance to the escape of fluid, that at any rate the slight increase of the intra-ocular pressure could not be augmented on account of the condition of the vessels. A series of sphygmographic tracings in these cases might, therefore, be interesting. Nettleship has already stated that the tension and organic changes in chronic cases may perhaps be sometimes due to feeble circulation or loss of arterial elasticity.

Again, if we suppose a slight increase of intra-ocular tension to have taken place, but that this does not balance the vascular tension so as to stop secretion from the vessels, we must expect a constantly increasing intra-ocular tension, or else admit that the conditions giving rise to the *plus* tension are always in simple glaucoma of a temporary or intermittent character.*

The acuity of the onset of symptoms would, therefore, appear to bear a direct relationship to the vascular tension in the uvea, or else the cause of their appearance must be of a rapidly fluctuating character. Once the passages are as completely and permanently blocked as they ever become, it is not unreasonable to suppose that after a period of specially high intra-ocular tension, due perhaps to a temporary increase in the vascular pressure, vessels usually given to passing fluid into the eye may take on the temporary role of carrying it away, the tension being greater on their exterior than in their interior. This power of course, could hold good only when such vessels continued to carry fluid, and not when that was interfered with, as by constriction of the emmissary veins.

One must bear in mind that it is easy to be mistaken in the condition of the anterior part of the eye. It is frequently said that because the anterior chamber appears to be of normal depth, there can be no apposition of cornea and iris. Now, the periphery of the iris and of the posterior surface of the cornea are quite invisible because they

*Examples of intermittency in ordinary subacute or chronic glaucoma are Bowman's case quoted by Priestley Smith and one by Randolph in the Annals of Ophthalmology, Vol. V, p. 945, in which the cornea exhibited a dense haze daily from 9 a. m. till 2 p. m., and in which the disease took more than four years to develop, V. of $^{20}/_{30}$ being then retained after iridectomy.

lie so far back, and adhesion of these two is quite compatible with a deep anterior chamber, whose depth may be increased, too, by a sudden backward bend of the iris at the point at which its corneal attachment ceases. (Treacher Collins 6). The alleged normal character of the anterior chamber is not susceptible of proof, because eyes affected with mere simple glaucoma are not excised, and even in the rare cases in which the filtration angle is found open in glaucoma, signs are not infrequently present that its patency is due to the excision of the globe.

Another argument in favor of these cases being true glaucoma is the growing feeling among ophthalmic surgeons that iridectomy is the most satisfactory treatment.

Prognosis.

While in the days before iridectomy the prognosis was almost invariably bad (*e.g.* Desmarres 7), at the present day, thanks to modern methods of diagnosis and treatment, a much more hopeful prospect can frequently be entertained. To go fully into this matter would be merely to repeat much of what has been already written, and a short consideration of some of the more important points in idiopathic glaucoma by which one may be guided must suffice. Nettleship (8) asserted that the occasional extreme chronicity of glaucoma precludes us from "saying with certainty whether the disease ever really stops short of blindness. Such arrest of progress, however, seems now and then to occur in the acute form."

In the acute form by operation we expect to remove so much of the blindness as depends on glaucomatous opacity of the media, and on temporary paralysis of visual nerve structure. The destruction of the latter of course depends upon the severity and duration of the attack, but one should be careful in prophecy, although generally a great deal may be expected from immediate treatment in moderately acute cases of short duration. The actual permanent cure of the disease will result only where a proper and sufficient circulation of the ocular fluids can be reproduced. In cases in which the symptoms are apparently not suscepitible of speedy relief by reduction of the tension, that is to say in the more chronic forms where the visual deficiency is due to lesions of a more permanent nature affecting the intra-ocular structures both anterior and posterior, the surgeon is usually well satisfied if his endeavors succeed in preventing further destruction to the

eye, and more than satisfied if they actually improve its seeing powers with the help of the necessary cylinders. The idea is very prevalent that operation, and especially iridectomy, not infrequently in chronic cases with contracted field is quickly followed by a still further contraction which may involve the macula. As early as 1862, v. Graefe (9) pointed out this danger with special reference to myopic eyes, and repeated the observation later (10). Berlin (11) in 1869 reported five cases of simple glaucoma, not myopic, in which just after the operation rapid diminution of the field occurred; and Mauthner, who also quotes the above, at the same time reported a similar experience. The reality of this danger was discussed at the meeting of the British Medical Association in 1895. (See under treatment). Nettleship (12) has found the action of the pupil under eserine to be the best prognostic guide in simple glaucoma, and in all of his successful cases before an iridectomy it reacted well to the miotic. This is explained, as Treacher Collins remarks (13), by the fact that its incapacity for movement is due to adhesion of the iris with the cornea; and when uveal ectropion is also present atrophy of the iritic tissue, the result of this adhesion, may be diagnosed as well. The condition of the field of vision, of the anterior chamber, of the excavation, and of the disc as regards color Nettleship considers to afford no constant guide as to the results of operative measures. "Thus we may say that senile cachexia in its various forms is distinctly unfavorable to operation in chronic glaucoma, and that absence of senility and an active pupil are favorable points." Nettleship expressed himself as having more faith in iridectomy in simple cases than before he had looked specially into his results. Stedman Bull (14) agreed with Nettleship's opinions, except that he thought that the latter had overrated somewhat the value of the condition of the iris as a prognostic guide. The effects of operation on 169 eyes with simple glaucoma were the following:

Vision improved for a time, but a few months later it and the field continued to decrease in	10 eyes
Vision remained unchanged for a year or longer in	36 eyes
Vision became slowly worse in	111 eyes
Vision became rapidly worse in	12 eyes

Knapp, criticising Bull's paper, stated that he was guided more by the iris than by the disc. He considered glaucoma to be a form of degeneration of the vascular

structures, evidenced by the condition of the vessels of the iris, and on that basis he has never been deceived in forming a prognosis. Others have written on this subject; as fair examples may be quoted v. Graefe, who found that in simple glaucoma iridectomy cured in fully half the number; in a quarter the disease recurred and was set aside by a second iridectomy; whilst in the remainder the eyes continued to go down hill.

Horner's statistics, given by Sulzer and quoted by Berry (14) from 1861 till 1881, are based upon 103 iridectomies for simple, and 149 for inflammatory glaucoma:

	INFLAMMATORY.	SIMPLE.
Improved	72.5 per cent.	22.3 per cent.
As before operation	11.5 " "	37.0 " "
Vision partly retained	10.0 " "	23.0 " "
Not improved and ultimately lost	6.0 " "	17.7 " "

Gruening (15) in 20 iridectomies for simple glaucoma had 4 eyes become blind, but in most of them the *status quo antea* was retained after many years. Fuchs (16) has found iridectomy cure 19 out of 39 of his own cases; *i. e.*, the vision either did not get worse or was actually improved, and in 20 the eyes in spite of the operation lost ground. These cases had all been followed for from five to ten years after the operation. An important consideration which should not be lost sight of is, that there has been no method of operating common to all surgeons, although without doubt in the vast majority of iridectomies the iris has been excised, which, as we have seen, is a much less certain means of opening up the filtration angle than the more recent procedure of iridectome-dialysis. Fuchs (17) states that in considering the chances of improvement he pays more attention to the color of the papilla and to the caliber of the retinal arteries than to the mere depth of the excavation, but does not speak of the pupil in this connection.

REFERENCES CONTAINED IN CHAPTER XII.

1. Schweigger—Arch. Oph. N. Y., 1891.
2. Somya—Klin. Monatsbl. f. Aug., XXXI, 1893, p. 390.
3. Brailey—Trans. Oph. Soc. of Gt. Brit., XIII, p. 63.
4. Bitzos—Ann. d' Oeul., CXII, 1894, p. 92.
5. Zentmayer and Posey—Arch. Oph., N. Y., XXIV, 1895.
6. Treacher Collins—R. L. O. H. Rep., XIII, p. 483.

7. Desmarres—"Traité théorique et praqtique des Maladies des Yeux," 1858, III, p. 739.
8. Nettleship—R. L. O. H. Rep., p. 72
9. v. Graefe—Arch. f. Oph., VIII, 3, p. 307.
10. v. Graefe—Arch. f. Oph., XV, 3, 1869, p. 182.
11. Berlin—Report of Heidelberg Con., 1869.
12. Nettleship—loc. cit., (8).
13. Treacher Collins—R. L. O. H. Rep., XIII, 1891.
14. Stedman Bull—Trans. Am. Oph. Soc., 1889.
15. Berry—"Diseases of the Eye," p. 222.
16. Gruening—Trans. Am. Oph. Soc., 1888-89, V. p. 287.
17. Fuchs—"Lehrb. d. Aug.," p. 404.
18. Ditto, p. 378.

CHAPTER XIII.

TREATMENT.

PREVENTION; INTERNAL REMEDIES; MYDRIATICS; MIOTICS; ELECTRICITY; OPERATIVE MEASURES; CURE BY SCLERAL PUNCTURE; BY THE SCLERAL INCISION OF IRIDECTOMY.

Preventive Treatment

Can anything be done to prevent the occurrence of glaucoma in persons supposed to be predisposed to it, but in whom no symptom has shown itself? As the disease is sometimes hereditary, Nettleship has advised that the eyes of children of glaucomatous patients should be examined for premonitory symptoms. But especially in advanced life, and more particularly where the size of the cornea is below the average, one should aim at the prevention as far as possible of local congestions by the use of proper glasses, good light, the avoidance of the stooping posture at work, and undue excitement, while a generally healthy condition of the body, and especially of the circulatory system, is maintained.

Internal Remedies.

Before operative measures were recognized as advantageous in glaucoma, treatment consisted in prescribing for constitutional idiosyncrasies, especially gout, attending to the state of the digestive and sexual organs, and the use of local antiphlogistics (Sichel 1), MacKenzie (2) recommended besides bleeding, counter-irritation to the shoulder, etc., careful hygiene, purging, calomel and opium (mercury being useful in deep-seated ocular disease,) anti-neuralgics (belladonna, aconite,) iron and quinine.

It is now recognized that sleep, and even ease of mind, may cut short an acute attack, and morphia, which also tends to contraction of the pupil, or chloral may often be beneficially prescribed, while derangements of the general health, as in all other complaints, should be carefully investigated and treated. The circulatory and digestive systems require special attention, and constipation should be avoided. To gout has long been ascribed an intimate connection with glaucoma, and for this reason anti-gout

diet and medicines have been advised by the upholders of this relationship. Richey (3), a firm believer in the hypothesis that glaucoma is gout in the eye, along with arsenic, lithium, salol or salicylate of soda, nitroglycerine, nitrite of amyl, ammonium, taraxacum, colchicum, calomel, nux vomica internally, uses hot baths, and locally eserine or pilocarpine, galvanism, and local massage. With some surgeons salicylate of soda is part of a routine treatment. Sutphen (4) has reported a number of cases without rheumatic history, in which this drug relieved the pain. Strychnine has sometimes been found to improve the visual functions of an optic nerve partially destroyed by glaucoma.

Mydriatics.

The employment of a mydriatic for a glaucomatous eye is generally due to carelessness or ignorance, and with the result of increasing the tension. In the time of MacKenzie it was, as he said, sometimes used in glaucoma to improve vision, but had frequently to be abandoned on account of increase of symptoms. It is rarely in these days that it is used by the skilled ophthalmologist in a recognized case of glaucoma, but there are occasions on which it may be employed with benefit. One of these is in secondary cases due to certain positions of a dislocated lens. Another may be when the tension is the result of the aqueous humor containing albumen and solid matters (see serous iritis) and it is doubtful whether it can do any harm in cases in which the pupillary edge of the iris is bound down to the lens so that the pupil is unable to dilate, though, for the same reason, the benefits of atropin as a local antiphlogistic would be considerably lessened, seeing that its action in iritis probably depends to some extent on the anæmia and muscular rest produced by full dilatation. Noyes (5) has said that he is acquainted with an ophthalmic surgeon who is astigmatic and glaucomatous, and who successfully treats the glaucoma, the nature of which is not stated, by suspending his accommodation by means of atropin and using full correction.

Miotics.*

Calabar bean, it was expected, would heighten the nor-

*This word is usually spelled "myotics," but, as has been pointed out by Hirschberg, "miotic" is correct, according to the Greek derivation.

mal tension, and it was found to do so by Adamuek, von Hippel and Gruenhagen, though this has not been borne out by subsequent observers. It had already been used by von Graefe to facilitate the operation of iridectomy by narrowing the pupil. It was used by Laqueur (6) in March, 1876, on the eyes of a woman of 48 years of age, with acute glaucoma, drops of the strength of one-third per cent. being employed with excellent result; and then on a case of secondary glaucoma, due to dislocation of the lens, the tension being reduced to normal in fifteen minutes. It was also employed by Adolph Weber (7) in glaucoma about the same time. In spite of his favorable impression of its effects in glaucomatous tension Laqueur (8) asserted that "physostigmine should in no way set aside iridectomy in acute glaucoma," and others fearing that by its use operation might be unduly postponed argued against even its occasional employment. Since Laqueur's time it is probable that his advice has not been followed so faithfully as it deserved, and that the treatment of glaucoma by miotics has assumed in some places a position more prominent than is wise. There is no doubt that these sometimes will cure, and especially in cases produced by the use of mydriatics or by operation on the other eye, as well as that they are most useful in softening the globe before operation, or to replace operation when for some reason that is contra-indicated. At the same time their substitution for iridectomy in chronic cases has frequently gone too far. Their beneficial action is generally supposed to be the result of the enlargement of the angle of the anterior chamber, due to the thinning of the iris on contraction of the pupil. Coincidentally the iris becomes more hyperæmic and the ciliary muscle is spasmodically contracted. It is therefore obvious that before their use the surgeon should assure himself of the absence of cyclitis or iritis, the latter with posterior synechia having even been produced by eserine in a glaucomatous eye (*e.g.* Gruening 9); and it is apparent also that miotics must be useless when the iris is atrophic, or where from any cause, such as peripheral, synechia in old glaucoma, it is prevented from retracting from the filtration angle. There even appear to be cases of acute or subacute glaucoma without atrophy of the iris, in which eserine has no effect as a miotic, whether or not on account of non-absorption is uncertain. The two miotics in general use are sulphate

or salicylate of eserine and nitrate or hydrochlorate of pilocarpine, some surgeons preferring the latter because, as they say, it acts less energetically. The more extreme advocates of pilocarpine sometimes assert that eserin will produce glaucoma where the former will not, and quote the fact that eserin solutions are apt to cloud with fungus, but equally experienced surgeons have found no fault with eserin which is not equally applicable to pilocarpin, and the matter seems to resolve itself into a question of the strength of the solution. Eserin is a much more active agent than pilocarpin, and should be correspondingly diluted. When either is used in a too concentrated form irritation is apt to be set up, and the usual dull pain experienced after its instillation may become excessive. Pilocarpine is employed in a strength of from 1 to 8 grains, and eserine of $\frac{1}{8}$ to 4 grains, to the ounce of water. Probably the most satisfactory way is to begin, as was recommended by Sir William Bowman, with a weak solution of say $\frac{1}{8}$ of a grain of eserine to the ounce, and increase the strength according to circumstances. Priestley Smith thinks 1 grain to the ounce of sulphate of eserine the strongest solution that is ever necessary, and advises the very satisfactory combination of 5 grains of hydrochlorate of cocaine in the ounce of this eserine solution, sufficient to diminish ciliary hyperæmia and excitability, while not strong enough to interfere with the desired miosis.

Electricity.

Dr. M. F. Pilgrim (11) has recorded 2 cases of acute senile glaucoma, and 1 case of chronic glaucoma in a lady of 29 years of age, in whom the use of a mild galvanic current, "the anode to the nape of the neck and the cathode covered with soft sponge over the affected eyeball, making gentle pressure and moving it too and fro," had an immediately beneficial effect on the tension and pain, and later on the vision. Reversing the current caused increase of pain in the young lady's case. During the subsequent treatment, and, indeed, before the galvanism in the acute cases, pilocarpine was used. The massage of the eyeball should also be noted. Dr. Pilgrim explains the action by quoting Dr. W. J. Morton on a "Possible Electric Polarity of Metabolism and its Relation to Electric Therapeutics and Electric Physiology," thus: All active centers of

chemical exchanges are electro-positive. When these are over active, an applied pole decreases the activity and cures the disease, opposite conditions producing the reverse effect. This is a not too modest claim for electro-therapeutics, and those who understand the present position of our knowledge of glaucoma will be chary of giving credence to the alleged powers of galvanism over it, especially in chronic cases.

Richey has also treated simple glaucoma (12) by the application of electricity to the cervical ganglion and the fifth nerve, on the supposition that glaucoma is a vascular disease.

Operations.

It is long since operative measures began to be employed for the treatment of glaucoma. Simple puncture of some part of the globe, in order to diminish the excessive tension, was first tried. Thus MacKenzie (13) employed

Paracentesis of the Cornea,

and found that it sometimes gave transient relief from pain and improvement in vision. Desmarres (14) also had the same experience, and found the beneficial results less lasting than those of puncture through the sclera—frequently only about four hours. v. Graefe in 1857 (15) had observed only two cases in which permanent benefit resulted from the operation. Others had a similar experience. In the high tension resulting from "serous iritis," paracentesis may frequently be employed with the best results. MacKenzie also tried

Puncture of the Sclera,

without lasting benefit. It might be done in front of the iris, "anterior sclerotomy," or behind it, passing through the choroid, "posterior sclerotomy." For the latter he recommended that a broad iris knife, pushed through the sclerotic "toward the center of the vitreous humor, is to be turned a little on its axis and held for a minute or two in the same position, so that the fluid may escape." Other surgeons had continued occasionally to employ it, but usually with only temporary benefit, until it recently came again into prominence. Mauthner (16) in 1878, speaking of "the puncture of the sclerotic with a needle," quoted Middleton, 1835, Richey, 1853, Leport, 1876, and said that Hancock in 1860, made his sclerotomy

with a knife or needle through the ciliary muscle. At the ophthalmological congress held in Edinburgh in 1894, Dr. Parent of Paris, said that in France this operation is named after Leport, and Mr. Power claimed it for the English surgeon, Hancock. While it would seem from the above that neither is properly entitled to any honor which may be attached to priority in its use, its value is mainly as an adjunct to iridectomy in cases of malignant glaucoma Weber (17,) and Nicati (18,) or as a preliminary to facilitate this operation. Priestley Smith (19) in 1894, stated that he had already done 50 such preliminary operations without any evil result. He punctures the sclera with a Graefe knife 5 m.m. only from the outer margin of the cornea, so as to open the vitreous chamber as far forward as possible. The knife is directed toward the center of the globe in order to avoid the lens, and during its withdrawal it is rotated on its axis so as to make an L shaped aperture, more favorable for leakage than is a linear cut. Pressure is applied to the cornea for some twenty seconds or more, to force the lens back and to open the angle. In this way the anterior chamber is deepened and the incision therefore, facilitated, while the dangers of intra-ocular hemorrhage from a more sudden diminution of the intra-ocular tension are no doubt lessened. One may perhaps also find some difficulty in reconciling the generally immedia ely good effects of iridectomy on a glaucoma originating, according to the theory with which Priestley Smith's name is chiefly associatec, behind the lento-zonular diaphragm; whereas a preliminary posterior sclerotomy with pressure on the cornea will tend to replace the lens and raise the blockade in a manner agreeable to this theory, which he has so laboriously and so successfully worked out. Other surgeons who have adopted this operation, have not all thought it well to incise so far forward; in the writer's experience, a distance of 8 or 10 m.m. from the cornea, as Weber advised, being preferred as less likely to injure the ciliary body. In selecting a position it is well to avoid as far as possible, the vicinity of the recti muscles, close to which, as the writer (20) has observed, the vortex veins are generally to be found piercing the sclera at an average distance of 13 m.m. behind the corneal margin.

The beneficial action of a preliminary posterior sclerotomy in difficult cases of iridectomy is confirmed by Nicati (21) and Gifford (22), as well as by Abadie, Pflueger, Mac-

Hardie, etc., at the Edinburgh International Ophthalmic Congress, in 1894. Nicati (23) uses "equatorial sclerotomy" to re-establish the anterior chamber, etc., when necessary.

In spite of the harmless character which has been given to this operation by its advocates, one at least of the dangers which must occur to every one as a possible sequela, has once been found as a reality by Treacher Collins (24). Suppuration followed the incision and the eye had to be removed. "Sections across the line of puncture show a prolapse of the vitreous—much infiltration, with pus cells lying in it. It must have been along this prolapse of vitreous that the suppurative infection had gained access to the interior of the eye."

Iridectomy.

The discovery of the value of iridectomy, whether as some assert, by a happy chance, or as a result of reasoning from observation of its effects on other cases, may be left, even alone, to keep v. Graefe's name forever prominent in the annals of ophthalmic science. He wrote in 1856 (25) that four years previously he first did the operation of coremorphosis, or the production of an artificial pupil, in a case of recurrent iritis with occluded pupil and bulging forward of the iris, complicated by choroiditis, the posterior synechia being, he thought, the cause of the recurrences. The return of good vision he put down to improvement in the choroid. In 1857 (26), when he found paracentesis gave only a temporary effect, he began to think of some way to reduce tension permanently. Just about this time he was doing iridectomy for irido-choroiditis. He said, "it was chiefly the uses of iridectomy in ulceration and infiltration of the cornea which led up to my use of it for lowering the tension," but he had also noted its beneficial effects in staphylomatous eyes, which frequently are glaucomatous in their tension. He first actually tried iridectomy in glaucoma in June, 1856 (27). In 1858, Desmarres (28) writing on glaucoma, stated it to be incurable, but observed that v. Graefe had addressed a note in 1857 to the Institute of France, on the subject of iridectomy. "If these researches," he said, "are confirmed, this will be certainly an important application of iridectomy," but he doubted if their results would be more lasting than those of puncture of the sclera. In 1862

Bowman (29) asserted that some ophthalmic surgeons had not yet been converted to a belief in the use of iridectomy in glaucoma; but in the time that has elapsed since then the smooth current of its popularity has been ruffled only by the question as to the precise means by which its beneficial action is produced, which for a time in the hands of some surgeons gave it a place secondary to sclerotomy, but which has proved of ultimate value in stimulating research into the nature and etiology of the disease. The answers to this question of its action, and to a less extent to the other, whether it can ever be advantageously replaced by any different operation, must occupy a large proportion of what has to be said concerning it.

Benefits of Iridectomy Due to the Scleral Incision.

Something like a decade after v. Graefe's introduction of iridectomy for glaucoma, the necessity of interfering with the iris at all began to be questioned by Stellwag and Wecker, with the result that they both concluded that the important part of the operation lies in the incision in the sclera, though they differed concerning its mode of action. Stellwag (30) first reported the cure of glaucoma by sclerotomy without excision of the iris—two cases— which he explained on the theory that *the new cicatricial tissue stretched itself* according to the intra-ocular tension, which could, therefore, not become excessive. as evidence of which he claimed to have cured a case of glaucoma by merely incising one-third of the thickness of the sclera, (31). To this certain surgeons gave their adherence, among whom were Stilling and v. Hasner. Wecker (32), who had two years previously expressed his opinion that if sclerotomy could be performed without prolapse of the iris the results would be as good as those of iridectomy, at the Heidelberg Congress in 1869 again brought forward this idea. Later, in 1871, we find Stellwag (33), without having changed his ideas, declaring the necessity of excision of the iris, but only to prevent it from prolapsing. In the same year two steps forward of importance were made; one by Quaglino, and the other by Wecker. The former (34) reported 5 cases of the operation, and based his practice on the supposition resembling that of Stellwag, that by it the capacity of the globe was increased, and later, before incising, which he did 2 m.m. behind the

corneo-scleral junction, first began the local use of calabar bean to prevent prolapse of the iris, the important step referred to. Wecker, in the mean time, besides reporting on the subject to the Heidelberg meeting of 1871, had perfected the operation by leaving a bridge of uncut tissue which, with the aid of calabar bean, greatly enhanced the chance of healing without prolapse of the iris, his chief contribution to the success of the operation. By the year 1873 he had concluded, however, that iridectomy gave better results than sclerotomy, except in cases of absolute glaucoma, or where the iris is too rotten to remove: and the latter fell into comparative disuse in his hands until, under the influence of an idea that *the cicatrix acted as a filter* for the removal of excessive aqueous humor, he was again using it in 1877 (35), but only to once more renounce it except for absolute and hemorrhagic cases (36). Bader (37) in 1876, did sclerotomy with the object of forming a "permanent communication between the aqueous chamber and the subconjunctival space adjoining the scleral wound," which included a third of the whole circumference, the bridge left being composed of cunjunctiva only. Bader himself said of his operation that its only troublesome sequela was occasional bulging of the conjunctiva, or of the conjunctiva and iris, but the impression conveyed by what literature there is on the subject, is that more or less prolapse of the iris was, to say the least of it, frequent. Hirschberg (38), who had seen the operation and its effects in London, condemned it in unmistakable terms; and Mauthner (39) called it a "monstrosity." Bader (40) in 1881 still operated in the same way in order to secure and maintain a staphyloma of the conjunctiva with or without prolapse of the iris. Schnabel (41) considered that if the mere section of the sclera cured glaucoma at all it did so only by acting as a neurotomy; and later, while admitting that Wecker's "filtration loop" acts just as efficiently in reducing tension, as does either excision of the iris or a mere scleral incision, asserted that these latter are not beneficial as is the first, by forming a new outlet for the pent up fluid, a double corneal fistula. Knies had now, 1878, adopted the theory that iridectomy acted on account of the scleral wound, the removal of the iris being useful only to prevent the occurrence of its prolapse, a case which he published (42) in which the tension became and remained normal, while a corneo-iritic adhesion continued,

satisfying him that the aqueous fluid must have found a new exit through the wound. Mauthner, to whose retrospect on the subject of sclerotomy the writer is in part indebted for some of the above details (43), was also, on the whole, a believer in the efficacy of the scleral incision, as opposed to the excision of the iris, though he considered that the proper explanation of the action of iridectomy had probably yet to be discovered. Sclerotomy, he said, acted "just the same as iridectomy," and should be employed in the prodromal stage in glaucoma simplex early, in chronic glaucoma where there is a defect of the field near the fixation point, and in congenital hydrophthalmos, as well as in glaucoma from dislocation of the lens, glaucoma hæmorrhagicum, and glaucoma absolutum. Brailey, in 1881, gave it as his opinion that (44) when the iris is too adherent to the cornea to be removed, sclerotomy, where it is effective, acts by making a new *access to Schlemm's canal*. Nettleship, in 1888, (45) wrote: "So much of the future as depends upon the maintenance of normal tension will be distinctly favored by a porous scar; but, if the patient be old, and his tissues degenerated, such a scar may act unfavorably by increasing the likelihood of slow inflammatory processes," and possibly by acute infective inflammation. Snellen (46) wrote: "Should it be proved, as I think it will, that the essential part of the operation is the scleral wound, it will be necessary in many cases to combine an iridectomy with it, because when the pressure is high the iris very easily prolapses, and is apt to check the free escape of fluid from the chamber. Accurate measurements of the corneal curvature before and after sclerotomy and iridectomy, have shown that a certain flattening of the cornea results from the operation, especially when the eye is hard. This flattening cannot occur except as a result of an ectasia at the margin of the sclera, and at the same time the ciliary body must be removed from its previous contact with the lens by this yielding of the sclera. In my opinion, the effect of our glaucoma operations depends on *restoring the perilenticular space* through the anterior ectasia of the outer coat of the eye." Priestley Smith's (47) experience has led him to believe that Wecker's explanation of the action of iridectomy, the formation of a permanent filtration cicatrix, or the establishment of communication with the veins, holds true in a very considerable number of cases. "The lips of the

wound do not come into close apposition, and the overlying conjunctiva remains slightly elevated by the fluid which collects beneath it." Sometimes a visible extrusion of fluid into the conjunctiva can be produced weeks after the operation by pressure with the finger, with a consequent slackening of the globe, so much so, that Dianoux has recommended its systematic employment after sclerotomy, (48). Speaking on this subject in 1893, Koenigstein (49) says: "When a small cystoid cicatrix exists, massage of the eye drives fluid out visibly, and some authors advise massage, and some do it before the operation, but this is harmful in acute cases. Others even intentionally let the iris heal in the wound (cicatrisotomy)." Priestley Smith has examined one eye cured of glaucoma by operation, but excised twenty years later for pain, redness, and tenderness, without increase of tension, of 2 years' duration. He found that the wounded surfaces had united incompletely, that iris tissue was included between them in places, and that there were small open channels passing completely through the corneo-sclera, and leading into an open space beneath the thickened and elevated conjunctiva. This form of cure, he thinks, is most conspicuously present where the operation has been performed late, and in which a complete restitution of the filtration angle can hardly be expected. "Moreover, the fistulous condition, when strongly marked, appears to expose the eye to some additional risk from septic inflammation in the future." Then he says that the success of sclerotomy in many cases "proves that the essential part of an iridectomy is the incision of the sclera, not the excision of the iris; it does not prove, however, that the latter step is better omitted." In 1894, (50) Priestley Smith remarked that "it is generally recognized that the efficiency of a glaucoma iridectomy depends essentially upon the corneo-scleral incision, and that the removal of a portion of the iris is necessary only for the prevention of subsequent complications," and in July of the same year (51) speaking of an operation on a young patient, he said, that its result, "so far in the present case is a satisfactory filtration scar, with a high degree of astigmatism which was not present before the operation, and which depends on flattening of the cornea in a vertical meridian. It is reasonable to suppose that this change of shape has increased the distance between Schlemm's canal and the margin of

the lens in the region of the coloboma, and that although the lens will presumably still enlarge as life advances, filtration may go on at this part of the circle for many years to come." Treacher Collins (52) pointed out that a cystoid cicatrix is always lined by a more or less atrophied iris tissue, a fold of which had prolapsed into the wound. v. Graefe showed that these cicatrices periodically discharge themselves subconjunctivally. In one of the eyes which Collins was able to examine which had been cured of glaucoma by operation, he found that the ciliary body and atrophied iris occupied the gap in the corneo-scleral tissue, and that the tension had been reduced, as he considered, by this new channel of escape for the fluid. Sclerotomy is efficient as a reducer of tension (a) in such a condition, the iris tissue becoming permeable as it atrophies; (b) when by the escape of the aqueous the iris is displaced from its apposition with the cornea; (c) when a permanent gap is formed in the adherent root of the iris through which the aqueous passes directly from the posterior chamber to the filtration area.

While it must be admitted that the evidence brought forward in the above arguments is sufficient to show that in all probability it is the scleral incision which cures glaucoma in a certain number of cases, care should be taken to distinguish a wound which has united firmly without the inclusion of iris or ciliary processes from one in which part of the uvea is incorporated. It is in the latter case only that we can count it as satisfactorily established that the cicatrix plays the principal role, producing however, at the same time, a condition of things by no means devoid of future danger to the eye. In the former case, Snellen's idea of the enlarged circumlental space fits in with such theories of the etiology of glaucoma as represent its origin from an obstruction in that region. If, however, during the operation the iris and cornea be separated, and ciliary congestion reduced, we have probably in these as good an explanation of reduced tension; if the iris and cornea be not separated, the mere permeability of the perilenticular space will not suffice to permit of filtration from the angle, and if the aqueous continue to possess an exit in the scar, through either the anterior or the posterior chamber, that alone might be sufficient to account for the non-return of the glaucoma. It appears to the writer very doubtful that the scleral incision causes at,

or soon after the time of the operation, any bulging likely to open up the perilenticular space, and Priestley Smith's practice of a preliminary posterior sclerotomy appears to forward this view. But if the bulging, which does frequently occur later, affect the sclera so far back as the level of a possible lento-ciliary obstruction, it may in that case very well account, to the minds of those who agree with Priestley Smith in his theory of glaucoma, for the immunity of many eyes operated upon from recurrence of high tension, but only if combined with an open filtration angle, or a leaking wound. Alteration in the shape of the cornea in front of the wound does not, of course, necessarily imply alteration in the shape of the sclera behind it. Along with the arguments advanced in favor of the important action of the scleral incision, the following should be considered: v. Graefe (53), who relinquished his own theory that iridectomy acted by diminishing the secreting surface, wrote in 1869, "the most unfortunate of all (theories is) that which refers the effect simply to the wound in the capsule of the globe." Del Monte (54), and Laqueur (55), have seen iridectomy control glaucoma, when the incision has been entirely in the cornea. Schweigger (56) said that even the most peripheral wound lies chiefly in the cornea, that it heals by direct union, and not as Stellwag asserted, with interposition of new tissue, and Quaglino, at the International Congress in London, in 1872, was no longer able to support Stellwag's hypothesis. Wecker, as we have already seen, found after various trials, that sclerotomy was, in practice, inferior to iridectomy. Schnabel (57) wrote, "the scleral cicatrix then possesses no peculiarity of structure by which its reputed function as a filtration tissue could be accounted for" He also asserted that v. Stellwag's opinion, that the cicatrix in the sclera is of loose connective tissue, is wrong. It is neither excision of the iris nor the scleral wound which cures, but a third unknown factor. He performed sclerotomy on one eye and iridectomy on the other, in six cases, and in only one did sclerotomy have as good a result as iridectomy, but this one shows that it is not necessary to touch the vascular tissues. Schoeler (58) showed that in rabbits' eyes, upon which sclerotomies have been performed, filtration is retarded, and not promoted, and that when the intra-ocular pressure was increased in these eyes, the cicatrices remained dry. But Priestley Smith's (59) contention inval-

idates this argument when applied to eyes with closed filtration angles which, unlike the healthy eye, may possess no other exit for the fluids besides the wound. Nettleship (60) returned to iridectomy in simple glaucoma after a tolerably extensive trial of sclerotomy, fifty cases at least. The results of the latter were often at first good, but later the eyes went down hill. Besides, the operation (he always employed Wecker's) is more difficult of performance. Treacher Collins (61) showed that many so-called scleral incisions lie really as much in the cornea as in the sclera. He pointed out that from the corneo-scleral junction in front at the termination of Bowman's membrane to a point on the globe level with Schlemm's canal the distance is 1.61 m.m., and to the commencement of the ligamentum pectinatum where Descemet's membrane splits it is from the same point 1 m.m. Collins considers it unlikely that the fibrous tissue of a scar should filter any more than the tissues of the cornea and sclera, and "pathologically," he says: "I find in eyes iridectomised for glaucoma good bands of cicatricial tissue, well at the periphery of the chamber, free, for some part of their extent, from any entanglement of iris, where *plus* tension returned and persisted." The object of some surgeons in operating by sclerotomy, is to open up Schlemm's canal. Treacher Collins, in 23 eyes, found that in only 2 had the operation touched the ligamentum pectinatum (in one of which the lens escaped and the ciliary body prolapsed next day), and in none was the canal of Schlemm reached. Most of the incisions passed through both cornea and sclera. Vincentiis (62) says that Wecker's theory of the filtering power of the scar has not been demonstrated, even clinically, and that it rests entirely on conjecture. Besides the above, the various arguments in favor of one or other of the methods of cure said to be dependent upon the removal of the iris must be included in our survey of the subject, as well as the fact of the almost universal preference for iridectomy in by far the greatest number of cases, even in the presence of an iris contracting efficiently to to eserin.

REFERENCES CONTAINED IN CHAPTER XIII.

1. Sichel—"Mémoire sur le Glaucome," Ann. d'ocul., 1841, 2. vol. V, p. 25, etc.
2. MacKenzie—"Diseases of the Eye," 1854.
3. Richey—Ann. Oph. & Otol., V, 4, p. 920.
4. Sutphen—Ann. Oph. Soc., 1895.
5. Noyes—Trans. Am. Med. Ass., V, 1889. p. 303.
6. Laqueur—Arch. f. Oph., XXIII, 1877, 3, p. 149.
7. Weber—Graefe's Arch., XXII., 4, p. 216.
8. Laqueur—Op. Cit., p. 169.
9. Gruening—Trans. Am. Med. Ass., 1889.
11. Pilgrim—Ann. Oph. &. Otol., April, 1895.
12. Discussion on Gruening's paper, Am. Oph. Soc., 1888-89, V, p. 303.
13. MacKenzie—"Diseases of the Eye," 1854.
14. Desmarres—"Traité théorique. et practique des Yeux, 1848.
15. v. Graefe—Arch. f. Oph. III, 1857, p. 490.
16. Mauthner—Arch. Oph., N. Y., 1878, p. 178.
17. Weber—Arch. Oph., XXIII, p. 86.
18. Nicati—Proc. of the Soc. de Chir., Paris, 1881.
19. Priestley Smith—"Glaucoma," p. 151, & Oph. Cong.Edin., 1894.
20. Stirling—R. L. O. H. Rep., XIII, 4, p. 419.
21. Nicati—Arch. d'Oph., 1890-1.
22. Gifford—Jour. Am. Med. Ass., XXI, 1893, p. 634.
23. Nicati—Oph. Rev., XIII., 1894. p. 126, & Rev. Gen., Jan., 1894, & Soc. de Chir., Paris, 1881.
24. T. Collins—Lancet, Dec., 1894.
25. v. Graefe—Arch. f. Oph., II, 2. p. 202.
26. v. Graefe—Arch. f. Oph., III, 3, p. 490.
27. v. Graefe—Arch. f. Oph., III, 2.
28. Desmarres—Op. Cit., 1858, III, pp. 737-739.
29. Bowman—Brit. Med. J., Oct. 11, 1862.
30. Stellwag—"Der Intra-oculare Druke," etc., Wien, 1868.
31. Koenigstein—Wien Med. Blaet., XVI, 1893, p. 17.
32. Wecker—Bericht d'Oph. Gesel. zu Heidel., 1869.
33. Stellwag—Lehrb. d'Pract. Aug., p. 356.
34. Quaglino—Ann. di ottal., I, p. 200.
35. Wecker—Ther. Ocul., I, p. 381.
36. Wecker—Klin. Monat. f. Augenh., May, 1878, p. 204.
37. Bader—R. L. O. H. Rep., VIII, 3, p. 430.
38. Hirschberg—Deutsch. Zeitsch. f. Prak. Med., 1877, p. 27.
39. Mauthner—Op. Cit., (16).
40. Bader—Trans. Inter. Med. Con., 1881, III, p. 98.
41. Schnabel—Op. Cit.
42. Knies—Arch. Oph., XXII, 3.
43. Mauthner—Op. Cit. (16).
44. Brailey—R. L. O. H. Rep., X, p. 282.
45. Nettleship—R. L. O. H. Rep., XII, p. 72.
46. Snellen—Oph. Rev., Feb., 1881, p. 48.
47. Priestley Smith—Glaucoma, p. 147.

48. Dianoux—Arch. d'ophth., Sept-Oct., 1883, p. 405. Oph. Rev., London, II, p. 304.
49. Koenigstein—Wien. Med. Blaet., XVI, 1893, p. 17.
50. Priestley Smith—Oph. Rev., Lon., Jan., 1894.
51. Ditto, July, 1894, p. 226.
52. Treacher Collins—R. L. O. H. Rep., XIII, 2, 1891.
53. v. Graefe—Arch. Oph., XV, 1869, 2, p. 253.
54. Del Monte—Naegele's Jahrsb., 1871, p. 276.
55. Ditto, p. 284.
56. Schweigger—Augenh., 1871.
57. Schnabel—Arch. Oph., N. Y., VII, 1878, p. 12.
58. Schoeler—Trans. Internat. Med. Con., 1881, IV. p. 100.
59. Priestley Smith—Glaucoma, p. 148.
60. Nettleship—Op. Cit., (45).
61. Treacher Collins—R. L. O. H. Rep., XIII, 2, 1891.
62. Vincentiis—Rev. Gen. d'oph., Nov. 30, 1894, p. 1.

CHAPTER XIV.

TREATMENT—(Continued).

GLAUCOMA CURED BY REMOVAL OF THE IRIS TO ITS UTMOST PERIPHERY; BY EXCISION OF THE SPHINCTER IRIDIS; BY MODIFYING THE CIRCULATION IN THE IRIS; BY DIVISION OF THE IRITIC VESSELS; BY THE PRODUCTION OF AN ADVANCED POSITION OF THE CILIARY PROCESSES; BY A COMBINATION OF AGENCIES. UNSUCCESSFUL IRIDECTOMY AND SCLEROTOMY. TREATMENT OF BUPHTHALMIA; OF GLAUCOMA IN APHAKIA; OF HÆMORRHAGIC GLAUCOMA; OF GLAUCOMA SECONDARY TO DISLOCATION OF LENS; OF GLAUCOMA SECONDARY TO SYNECHIA; OF SEROUS IRITIS; AND OF SIMPLE GLAUCOMA.

Removal of Iris to Periphery.

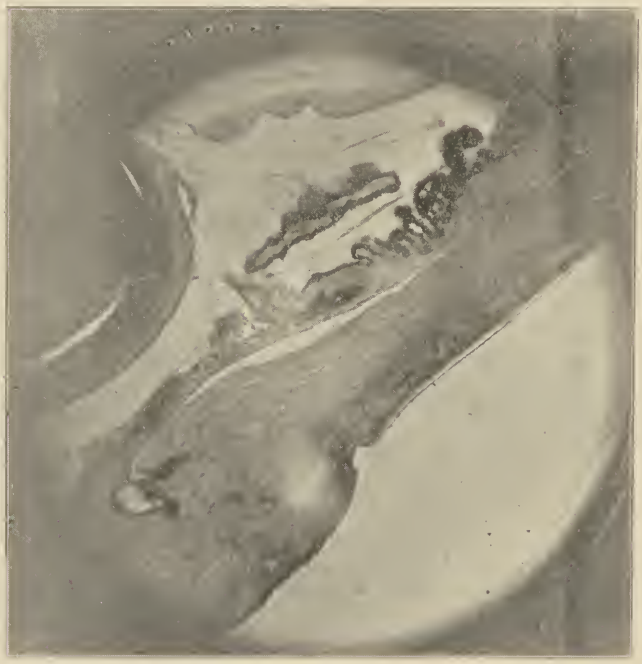

Fig. 12. Return of *plus* Tension after Iridectomy. The root of the iris was very adherent to the cornea, and the operation was not successful in separating them.

The logical sequence of views on the etiology of glaucoma, which consider the apposition of the iris and cornea

as an important factor in its production, is that for its cure that part of the iris liable to block the filtration angle should be, if possible, removed. As one would expect from Weber's ideas on the subject (1), he advised that the iris excision should be made for this purpose as peripheral as possible. Brailey (2) thought in 1881 that iridectomy acts by removing the periphery of the iris, and is effective provided that the inflammation is not in an acute stage. Treacher Collins (3) said: "In some other recent and acute cases I have shown that the iris tears away from its extreme root, thus leaving a large portion of the filtration area free for drainage, even should the remainder of the iris retain its faulty position." In two of the four cases cured by iridectomy which he had examined this seemed to be the method of cure, and the mere traction on the iris, or the escape of aqueous may suffice to separate iris and cornea, as evidently occurred in one of his specimens, an acute glaucoma of four days' standing, in which the angle was found open, but with a piece of iris left adhering to the cornea. Vincentiis (4) believes that in performing iridectomy there are two points of special importance; first, the traction exercised on the iris all round it, which effects its base where attached to the cornea; and second, the unfolding of the tissue of the iritic angle. The pressure with the scissors and the traction on the iris are a great strain on the iritic tissue and its connections. This traction and unfolding of the iris will avail only if the section of the iris grasped by the forceps preserves the capacity of transmitting force to the rest of the iris, which cannot happen if it is atrophic. While in old standing cases of glaucoma a cure can comparatively seldom be effected by the above means, the iris and cornea being so frequently inseparably united, it should yet always be the object of the surgeon to remove the iris up to its junction with the ciliary body, which adds another and in recent cases the best chance of benefiting the eye. Treacher Collins has shown that neglect of this important modification of iridectomy has been responsible in many cases for the non-success of the operation. Schnabel (5) did not believe that the operation acted in this way, because he had examined eyes recovered after iridectomy where the iris still remained peripherally attached to the cornea. In others it had been separated by traction at the operation, and in some it had become attached again, and

he quoted Knies's case, in which it was known that the tension became normal after an iridectomy which left the obnoxious synechia (6). In answer to Schnabel's objection it may be stated that the above is not claimed as the only method by which glaucoma may be cured, and that these cases were probably benefited by one of the others.

Excision of the Sphincter Iridis.

Von Graefe remained dissatisfied with all explanations of the cure by iridectomy which were given in his time. Yet he wrote (7): "The more sudden the cloudiness of the media with which the disease comes on, the more does the real point of the operation seem to lie in the excision of the sphincter;" and Schweigger (8) said: "In some cases of glaucoma that come for treatment on account of inflammatory symptoms, it is sufficient, as v. Graefe has mentioned, to excise a portion of the sphincter iridis." The proper explanation of this lies, most likely, not in the excision of part of the sphincter, but in the traction applied to the base of the iris, or in the effect of the scleral wound. Delmonte (9), Laqueur (10), Schnabel (11), and Mauthner (12) have seen iridectomy control glaucoma when the sphincter was left intact.

Modification of the Circulation in the Iris.

Some surgeons hold that in some way, concerning which there is little agreement, the removal of a piece of the iris modifies its circulation, and in a manner just as doubtful cures the glaucomatous tension, as v. Graefe had shown it could cure the diminished tension of some cases of irido-cyclitis. Exner asserted that at the edge of the coloboma arteries set up a novel connection directly with the veins, without the interposition of capillaries, which moderates the blood pressure in the iris, and this Schweigger (13) characterized as the only fact of any value relating to the action of iridectomy in glaucoma, a criticism with which he will find few surgeons to agree.

Berry wrote (14): "All that can be said then is that in some way or other, and by reason probably of the changes which the excision of a portion of the iris produces in the circulation in the iris, the operation of iridectomy is capable of effecting an improvement in the conditions of the

circulation in the ciliary body and the anterior part of the choroid."

Division of Iritic Vessels.

Brailey (15) suggested, as a result of his observations concerning the enlarged size of the *circulus arteriosus major*, that iridectomy may possibly cure "by *tearing or removing the base of the iris near a dilated vessel*, and that it fails when the tension depends on other causes."

Production of an Advanced Position of the Ciliary Processes.

Schnabel (16) thought that the value of iridectomy might perhaps lie in a forward traction of the ciliary processes, and their permanent retention in that position, but the writer is unable to explain in what manner he considered the benefit to arise.

A Combination of Agencies.

Koenigstein (17) took safe ground and said that iridectomy lessens the secretive surface, divides the nerves, brings the anterior chamber and the vitreous into closer relationship, completely removes the aqueous, dispenses with part of the capillary circulation, and produces a direct connection between iritic arteries and veins, whereby blood pressure, and with it intra-ocular pressure, are lowered, and finally through the traction on the iris and its excision, which should be broad and deep, the angle is reopened. Priestley Smith (18), while in other places more definitely specifying the part of the operation of most importance, yet writes: "In the earlier stages of primary glaucoma, iridectomy seems to act in the following way: In the first place, the escape of aqueous humor and the simultaneous advance of the lens immediately reduce the pressure in both chambers. The obstructed circulation in the uveal tract is relieved by the removal of the pressure, and in many it is further relieved by a free escape of blood from the divided vessels of the iris. Fluid drains away through the wound for some hours, and the overfilled vitreous chamber is depleted. The ciliary processes recede, the pressure on the iris base is relieved, and as the anterior chamber reforms the filtration angle reopens. Iridectomy acts with greater certainty in acute congestive than in chronic non-congestive glaucoma, and it is effec-

tive chiefly in the earliest stages of the disease. The reason of this is clear. The pressure on the iris base is more likely to be removed when it is due to vascular congestion than when it depends upon more permanent causes, and the filtration angle is more likely to reopen when the closure is still recent than when it has lasted many days or weeks. In two eyes examined by Fuchs which had been permanently cured of acute glaucoma by iridectomy the filtration angle was patent throughout the whole circle."

Unsuccessful Iridectomy or Sclerotomy.

As a rule, immediately upon the escape of fluid from the section the tension goes down and relief comes to the eye. An unsuccessful operation may show itself as what is called "glaucoma malignum," in which the eye, immediately after the operation, remains hard, the anterior chamber does not reform, and blindness rapidly ensues with much pain. Schweigger (19) wrote: "When glaucoma malignum attacks one eye it follows iridectomy on the other eye, even when the other eye is not affected for years after the first. The cause of the unsatisfactory results of the operation must lie in some obscure structural anomaly inherent in the eye itself." These cases most commonly occur in the simple non-congestive type of the disease. Weber's assertion that the propulsion of the lens toward the cicatrix may interfere with the action of an iridectomy is amply confirmed. Nettleship (20) said that "malignant" cases are sometimes due to a weak suspensory ligament allowing the lens to become dislocated; and Priestley Smith (21), that "the lens may be wounded, swell, and block the wound. The uninjured lens may be driven forward by pressure from behind and so completely block the wound and annihilate the angle of the chamber, so that no fluid escapes from the eye after the first hour or two, and the tension is soon as high as, or higher than before;" while Treacher Collins (22) has shown that an operation may fail from adhesion of the lens to the sclerocorneal cicatrix, so that the lens is displaced forward, its upper border blocking the freshly opened up filtration area, or pushing the iris or ciliary processes forward. This is most liable to occur where the anterior chamber is slow to form, because an excess of plastic material is then thrown out. For the further treatment of these cases the

old procedure advised by Weber (23) has been found successful in some cases. (Priestley Smith 24, Nicati 25, etc). Also the lens may be extracted in its capsule, though, as Nettleship (26) observed, unsound intra-ocular vessels are apt to be associated with a weak suspensory ligament. This need not necessarily deter one from the operation, but should always indicate great care during its performance, and it is a question whether a preliminary posterior sclerotomy would not always be advisable. Priestley Smith (27) considered that when Weber's operation fails it will probably be justifiable to reopen the original wound or to make an even larger one, and to attempt to extract the lens through a peripheral opening in the capsule. A discission of the capsule may be subsequently necessary if it adheres to the wound. Glaucoma malignum is one of the cases in which Rheindorf (28) removes the lens, and subsequently passes the knife into the "vitreous depression." An intra-ocular hemorrhage immediately following the opening of the globe is no doubt at times the real origin of the forward displacement of the moveable parts of the eye. At the risk of a second hemorrhage it would be better to operate as above than to see the eye blinded without an attempt to save it. But without the onset of glaucoma malignum, iridectomy and sclerotomy are sometimes of no avail in permanently reducing tension and saving the eye. Thus Brailey (29), in four cases in which operation gave no relief, found in three of them the iris still attached to the cornea; and again, out of twenty cases in which iridectomy had been performed, in six he found the iris incarcerated in the wound, in eleven the iris still adherent to the cornea, and three had had subsequent severe iritis or cyclitis. Brailey remarked that of these operations, scarcely any of which had taken place at Moorfields, only one had been efficiently performed. Priestley Smith (30) has found that "iridectomy sometimes fails; it effects neither a reopening of the filtration angle, nor the formation of vicarious channels. The incision may be too far from the periphery of the chamber; a purely corneal wound is apt to close too quickly, and the resulting cicatrix is less extensible than one which lies largely in scleral tissue. Especially in cases of absolute glaucoma, where a reopening of the filtration angle is out of the question, is such an operation powerless for permanent good." Treacher Collins (31)

said that iridectomy or sclerotomy fails when certain already mentioned conditions are absent. Thus *(a)*: When the angle is not opened in making the incision, or drawing on the iris; *(b)*, or when the iris does not tear away at the extreme root because *cut* off, or because too adherent; *(c)*, or because no permanent gap leads out of the posterior chamber. "By far the commonest cause of failure would seem to be the non-removal of the iris up to its extreme periphery" (32). In acute cases the iris is only in apposition with the cornea, while in chronic cases these are adherent. "This is the difference between acute and chronic glaucoma, which renders the operation of iridectomy such a very satisfactory proceeding in the former, and so very unsatisfactory in the latter."

Treatment of Buphthalmia.

In considering what treatment to employ in a case of buphthalmia one must bear in mind the anatomical condition of the eye, chiefly the tenuity of its walls and the stretched and possibly ruptured state of the suspensory ligament, as well as the restlessness of young children. Sclerotomy, and still more iridectomy, are, therefore, attended by the dangers of loss of vitreous, collapse of the globe, intra-ocular hemorrhage, etc.; and operations which have passed off without accident, while sometimes attended with good results, have not by any means been always so, as is to be expected from what we have seen of the etiology of these cases. This is especially so with sclerotomy, or, as pointed out by Treacher Collins (33), when an iridectomy has failed to remove the iris up to its very periphery. With our present better knowledge of the disease early iridectomy will probably come again to the front, but the recent tendency has certainly been to relinquish all operative procedure except perhaps frequent paracenteses, and to substitute entirely the use of miotics.* In secondary cases one's hopes of improvement will of course depend upon how far one can remove the cause. If the glaucoma is dependent on posterior synechia an iridectomy before the iris and cornea have become too firmly united would give a good prospect of at least

*E. g. See Panas' book on "Diseases of the Eye," page 525; Snellen on "Treatment of Infantile Glaucoma Leading to Buphthalmos" (34), and Swanzy on "Diseases of the Eye," p. 318, etc.

the prevention of any increase in the destruction of the eye. If due to anterior synechia, one should consider whether it would be possible to divide the iris by means of Lang's twin knives, or to otherwise open up such a communication between the posterior chamber and the exits from the eye as would be effectual for its drainage, such as by iridectomy, sclerotomy, or one of the newer operations.

Treatment in Aphakia.

In Stoelting's case section through the capsule, two sclerotomies, one iridectomy, and tearing the capsule were all of no avail. One of Mittermaier's cases was cured by iridectomy, and eserin reduced the tension in two others. In Natanson's first group, in which the increase of tension came on during the after treatment, miotics proved of little use; while iridectomy had good results in two, slight in four, and none in one case; sclerotomy in one advanced case was of no use; and four of the eyes had to be excised. In his second group, in which the increase of tension appeared several years after the extraction, iridectomy was done in several cases, but only once with success. Treacher Collins (34) advises an endeavor to prevent such cases by encouraging rapid healing of the wound, by leaving little lens cortex behind, and when needling by the use of such an instrument as will block the aperture in the cornea so that the risk of capsule, etc., being carried into it by escaping aqueous may be prevented. Swanzy, no doubt as a result of Collins' investigations, advises (35) that after extraction the wound should be searched with a bent iris forceps from end to end for tags of capsule, etc., which might prove a source of danger after it had healed. Paracenteses, sclerotomies, curette evacuations, and suctions had been tried in the cases published by Collins, but unsuccessfully; and in only one had he (in 1890) seen the glaucoma permanently cured by operation, an iridectomy followed by incision in the lens capsule. Knapp, whose custom it is to extract by the simple method, and frequently to needle, later claimed to have cured all his ten cases, one by eserine and the others by iridectomy, normal vitreous escaping each time during the operation.

Treatment of Hemorrhagic Glaucoma.

The writer cannot do better than quote the conclusions

of Bourgon (36), who made an exhaustive study of the results of treatment in the recorded cases of hemorrhagic glaucoma, as these have not been modified, so far as he is aware, by any writer since.

The Results of Iridectomy.

"Of 28 operations in 24 patients, 16 patients have denied any amelioration from the operation, and enucleation has been effected 11 times. In 5 other cases it has been proposed, but has not been effected on accout of the refusal of the patient." * * * "As a résumé—of 24 patients, 8 alone have found cessation of pain, and 5 of these have had a visual acuity superior to that which they possessed at the moment of the operation; the result is certainly of small satisfaction, but it would appear still more deplorable if one considered Galezowski's case in which a relapse took place only 23 months after the operation, and if one remembered that most of the patients registered as benefited or cured have been observed for only a few months or weeks afterward." * * * "We conclude that iridectomy is an operation to be rejected finally and absolutely in glaucoma hemorrhagicum." *Sclerotomy* "produces results incomparably superior to those of iridectomy; 7 ameliorations in 13 patients represent more than 50 per cent. of successes. *Paracentesis* is useless, and should be proscribed. *Stretching external nasal nerves* (37). The literature on this subject is scanty, and the operation apparently of little service. *Equatorial sclerotomy.* Randolph advised iridectomy, followed by this operation. *Trephining sclera.* Spencer Watson reported one case which remained well for three months and then relapsed. *Sclerotomy with drainage with gold threads* is abandoned by De Wecker, its author. *Quadruple peri-corneal incision.* Galezowski reported one case not since inflamed, but the operation was condemned by De Wecker and Abadie. *Crucial sclerotomy.* Galezowski advised four incisions, the knife penetrating the sclera 3 or 4 m.m. from the cornea, and being pushed from behind forward for ½ a cm. In one case the operation was ineffectual, but in the second the pain was relieved during the three months in which the case was observed.

Conclusion.

(a) True hemorrhagic glaucoma gives a very bad local prognosis. *(b)* The ordinary operations for glaucoma, except

sclerotomy, are useless and painful, and ought to be abandoned. *(c)* The general prognosis being nearly as grave as the local, the ophthalmologist ought to attract the attention of the patient's ordinary medical attendant to his condition, and especially to his vascular system. *(d)* Treatment ought to be the following: In the hemorrhagic period take care of the general health, and remove congestion from the eye. In the period of confirmed glaucoma, the treatment should first be medicinal; therefore, now add miotics, moist heat, and injections of ergotin in the temple. Bourgon reports that narcotics have all been of no avail (38). Stilling recommended chloral to reduce the *plus* tension. If the above methods are not sufficiently beneficial, then resort must be had to surgery, which may be summed up in the word sclerotomy, simple or equatorial, and if this be unsuccessful, the surgeon should enucleate without trying any other operation.

Treatment of Glaucoma Secondary to Dislocation of the Lens.

In 1876, Laqueur (39) used calabar bean for a case of glaucoma secondary to dislocation of the lens, and reduced the tension to normal in fifteen minutes, and in 1879, Mauthner (40) said: "The conditional supporters of sclerotomy will probably prefer to try it rather than iridectomy in glaucoma after dislocation of the lens." But now one will treat each case according to the manner in which the high tension is being brought about. It will generally appear that the best course to pursue will be to remove the lens, but in deciding upon this step, one must bear in mind the probable loss of vitreous during the operation, on account of rupture of its anterior supports. The age of the patient should also be considered, in deciding the nature of the operation. If the lens lies in the vitreous one will have to decide whether to leave it alone, and perhaps use miotics, or to extract it with a scoop. When in the pupil, or even in the anterior chamber, eserin is likely to do harm by closing more firmly the lymph path to the angle, while a mydriatic by dilating the pupil and drawing the iris away from the lens may open this path, and even permit of the return of the lens to its normal position, on the patient's throwing back his head. Such a case the writer had the opportunity of watching for some months, the lens frequently coming forward into the anterior cham-

ber, but under a mydriatic, it always returned behind the iris, when the high tension disappeared. Ultimately it was removed after a specially severe glaucomatous attack, and with only a slight loss of vitreous. When it is desired to operate upon a case of forward dislocation, it may be well previously to contract the pupil behind the lens, and so prevent it from slipping back behind the iris. If the patient be young, it may be best to remove the lens by needling and subsequent evacuation by suction or the curette.

Treatment of Glaucoma Due to Synechia.

The object of treatment in these cases is to open up the natural exits for the fluid, or to form a new one. This may be done by dividing a narrow tag approximating the cornea to the structures behind it; by making an aperture in an iris extensively adherent in front or behind, and opening up the filtration angle; or by making such an opening through the hull of the eye and the adherent base of the iris into the posterior chamber as will allow a permanent filtration without the necessity of an open passage through the anterior chamber.

Treatment of Serous Iritis.

As might be expected, eserin is usually of little value, and may do positive mischief in "serous iritis." Atropin is sometimes useful from its tendency to diminish the inflammatory state of the ciliary body; but along with mercurials or other internal remedies, tapping of the anterior chamber, which may require to be frequently repeated, will diminish tension and, therefore, the risks to the posterior nervous structures, and remove at least some of the deleterious matters which block the exits; for though the new fluid formed to replace that lost may contain much fibrin, the proportion of solids will probably decrease after each paracentesis. Where these have failed, an iridectomy sometimes has succeeded.

Treatment of Simple Glaucoma.

Iridectomy, the most reliable treatment for acute cases, has been by no means unanimously approved for simple glaucoma. (See prognosis). The reasons for that are apparent—the fact that certain surgeons are in doubt whether such are really cases of glaucoma at all, and still more, because the symptoms being insidious and little marked,

eyes are apt to have undergone such pathological changes before the operation as render their tissues little susceptible to improvement, either in position or texture. The comparative failure of iridectomy has, therefore, given rise to a large body of opinion that such cases are better treated by miotics alone, or in combination with internal remedies (see paragraphs on internal remedies); while many surgeons, considering sclerotomy a milder operation, and one followed less frequently by diminution in visual power, prefer it to iridectomy. For example, Noyes (41) preferred to leave cases of simple glaucoma alone, though he sometimes operated, and Galezowski (42) thought that sclerotomy, even if it have to be repeated more than once, is better than iridectomy. Abadie (43) in the slowly but continuously progressive forms of simple glaucoma advises, instead of operation, the administration of from 1 to 2 grammes of potassium bromide and half a gramme of sulphate of quinine in daily doses, along with the local instillation, once daily, of 2 drops of a half per cent. solution of eserin. As soon as the tension has become normal the bromide and quinine should be left off, but a carefully sterilized solution of eserin should be continued indefinitely.

De Wecker, who holds that in *every* case of glaucoma there is increased tension, goes so far as to assert that cases in which iridectomy or sclerotomy prove harmful, are not really glaucoma at all (44). In cases with no rise of tension, "false glaucoma," or optic atrophy, operation is contra-indicated, but miotics will be beneficial by diminishing even the normal tension, and thus reducing the pressure on the optic papilla, weakened by the peripapillary neuritis, which by subsequent exudation-contraction produces the atrophic cup that so resembles that of glaucoma. Mercury and iodides should also be given internally. In doubtful cases and in true simple glaucoma, Wecker advises operation, and sclerotomy preferably, as causing less visual disturbance than iridectomy.

A strong feeling has for some time been growing among another body of surgeons that after all a proper iridectomy is the best founded treatment, and that their results bear this out. Thus, Nettleship (45), in 1888 observed that eserin very rarely stops the progress of chronic glaucoma, though it is valuable in lessening the frequency and severity of the premonitions which often for a long time precede

the establishment of the permanent disease, and in checking fluctuations and recurrent threatenings after operation, and found that, on investigating his own results from iridectomy, they proved better than he had expected. The earlier an operation is performed the better, but he has stopped the progress of several cases even at a comparatively late stage.*

Stedman Bull (46) advised iridectomy on the worse eye first, at as early a stage as possible, delay being permissible only when vision and the visual field can be frequently tested and a miotic used; and Knapp (in discussing Bull's paper) while he also as a general statement advised early operation, shrank from it "when vision is still good and the visual field for ordinary purposes perfect," for these are the cases in which malignant glaucoma has been specially common in his experience. Gruening (47) in 20 iridectomies did not find any subsequent rapid contraction of the visual field or vision. Four cases became blind in time, but in most the *status quo ante* was retained after many years.

Treacher Collins (48) advised iridectomy before the iris and cornea have become inseparable; and when that has taken place to a marked degree "a sclerotomy performed with the intention of making a permanent gap in the adherent root of the iris would be more likely to succeed." The new operations described by Nicati, Knies, and Vincentiis, should also be considered. The following are the conclusions arrived at by Zentmayer and Posey (49) in a study of 165 cases of simple glaucoma on the books of Norris and Oliver, in their clinics in Philadelphia: "As operative measures are always to be deprecated when other measures are equally valuable, eserin should be employed in all cases of the disease." If, in a month's time the fields are diminishing, iridectomy should be employed. If the field have improved, "there is reason to expect that a beneficial action will be exerted for ten months upon the extent of the field and fifteen upon the visual acuity.

*Nettleship stated that there is a form of mydriasis which may follow upon sclerotomy, and which has a nervo-muscular origin, and he asked: "Is there any intrinsic neural apparatus in the ciliary muscle which, when uncontrolled by higher centers, can cause extreme mydriasis?" such as that which occurred in one of his cases, in which the iris shrank almost completely out of view three weeks after optico-ciliary neurotomy performed after loss of vision due to hemorrhage between the sclera and choroid, with complete temporary insensibility of the globe.

After iridectomy is performed, there is ground to believe that the course of the disease will be checked for a period of eighteen months in 15 per cent. of the cases. Eserin is powerless in 20 per cent.; iridectomy in 10 per cent. of the cases. So that 10 per cent. of all cases of simple glaucoma will not be benefited, despite all therapeutic measures which may be employed."

At a meeting of the British Medical Association in London in 1895, Nettleship, in discussing simple glaucoma, asked whether it is true that iridectomy, performed when the visual field is already lost up to the center, if often followed quickly by a further loss of the visual field which engulfs the center, and thus seriously damages visual acuity? And if so, is it more to be expected when the previous contraction is concentric, than when it has the form of a sector? Nettleship himself had had very few cases of quiet glaucoma in which the visual field had been decreased by operation, and quite a number in which it had then remained undisturbed. "The risk of such further rapid loss of field as a consequence of iridectomy is probably greater *ceteris paribus* when the tension is much increased." Mayer (Paris), when deciding as to the advisability of operation in any individual case, did not consider the state of either the vision or the visual field. Priestley Smith (Birmingham) said that the optic nerve, like other nerves, having once begun to atrophy tends to further atrophy when the nutrition of the nervous system in general fails and, therefore, it is well to care for the general health. Little (Manchester) had not found that iridectomy further contracted the visual fields. Swanzy (Dublin) would not operate when the field was contracted to the fixation point. Frost (London) had found preliminary scleral puncture useful in preventing failure of vision as a result of operation. Nettleship's next question was, whether it was advisable to operate early in simple glaucoma, or for fear of doing harm, to wait? He advocated early operation, and unlike Schweigger, first on the better eye. Before the disease had advanced, the risk was less of displacement of the lens or of internal hemorrhage, the ciliary processes and iris were less likely to be atrophic, and the hope of re-establishing the angle, was greater; also, of course, more vision could be saved. Fuchs (Vienna) had given up operating where there was no increase of tension. Critchett (London), Priestley

Smith, Swanzy, and MacKinlay (London), believed in early operation. Nettleship then went further, and enquired whether it was not well to operate, even in the prodromal stage. He said: "There can hardly be a doubt that if iridectomy were commonly performed in the prodromal stage, the number of persons who become blind of glaucoma would be considerably lessened. The risk of operation carefully done, is very small; the prospects of immunity conferred by it, very great." He advised operation at the earliest opportunity, especially in the young, because he believed glaucoma in them to mean a tendency to weakness of the suspensory ligament and blood vessels. For this reason, he incised further forward in the young. To this rule there were possible exceptions, when a miotic acted efficiently, and the patient could use it properly, and report himself regularly to the surgeon, but it was with dread that he saw such patients drift; also sometimes in the very old, or in otherwise bad subjects. Priestley Smith advised operation at any stage, provided that the patient fully understood the prognosis. Little agreed with Nettleship, that the action of miotics was unsatisfactory. As regards the nature of the operation employed, Nettleship stated that he had, after a prolonged use of it, quite relinquished sclerotomy in favor of iridectomy, except as a second operation when the latter failed, and then he made a Wecker's incision opposite the coloboma. He had used Priestley Smith's preliminary puncture on one or two occasions with satisfactory results, and he had no fault to find with it. Fuchs, Mayer, and Gayet (Lyons) always did iridectomy. Gayet made an equatorial scleral puncture instead of iridectomy when the latter appeared dangerous. Critchett's desire was to produce a cystoid cicatrix. Priestley Smith considered that success depends upon the production of a permanent subconjunctival fistula, and before operation for the avoidance of internal hemorrhage attended to the health of the patient as regards sleep and the action of the bowels and kidneys. If necessary, he slackened the eye by a preliminary scleral puncture. Little always did iridectomy, reserving sclerotomy for cases in which the former failed, when he incised in the region of the coloboma. A cystoid cicatrix was desirable. Power (London) stated that a temporary case, due to mental worry, could be cured by eserin and the treatment of the general health. Panas (Paris) asserted

that miotics take the foremost place in treatment, but as their action lasts not more than five hours, they must be instilled at least four times a day. He preferred an ointment composed of vaseline, salicylate of eserin, and hydrochlorate of pilocarpine. When that did not suffice, he did sclerotomy, and reserved iridectomy for cases in which that also failed. Frost did not operate so much now as formerly, from a suspicion that that did not arrest the disease. Williams (Liverpool) said that as sclerotomy was simpler he preferred it. Walker (Liverpool) had long ago given up iridectomy in glaucoma, but endeavored to produce instead, a cystoid cicatrix by dissecting up a flap of conjunctiva, and tucking it through the wound caused by the incision of the sclerotomy into the anterior chamber, which he had found efficacious.

It will be observed that on the whole, the tendency of those present was toward a wider field for iridectomy, but it is to be hoped that in future, in justice to each operation, its nature may be distinctly stated, whether a simple iridectomy, or an iridectome-dialysis.

REFERENCES CONTAINED IN CHAPTER XIV.

1. Weber—Arch. f. Oph., XXIII, 1877, p. 1.
2. Brailey—R. L. O. H. Rep., X. 1881, p. 282.
3. T. Collins—R. L. O. H. Rep., XIII, 1891, Lancet, December 22, 1894.
4. Vincentiis—Rev. gen. d'oph., Nov. 13, 1894.
5. Schnabel—Arch. Oph. N. Y., VII, 1878, p. 12.
6. Knies—Arch. Oph., XXII, 3, p. 200.
7. v. Graefe—Arch. f. Oph., XV, 3, p. 113.
8. Schweigger—Arch. Oph. N. Y., XX, 1891, p. 475.
9. Del Monte—Jahresb., 1871, p. 276.
10. Laquenr—Ditto, p. 284.
11. Schnabel—Arch. f. Oph., V, p. 379.
12. Mauthner—Orch. Oph. N. Y., VII, 1878, p. 178.
13. Schweigger—Op. Cit (8).
14. Berry—"Diseases of the Eye," p. 229.
15. Brailey—R. L. O. H. Rep., IX, 1877, p. 199.
16. Schnabel—Op. Cit (11).
17. Koenigstein—Wien. Med. Blaet., XV, 1892, p. 773.
18. P. Smith—Glaucoma, p. 145.
19. Schweigger—Op. Cit (8).
20. Nettleship—Op. Cit.
21. P. Smith—Glaucoma, p. 153.
22. T. Collins—R. L. O. H. Rep., XIII, 2.
23. Weber—Arch. Oph., XXIII, 1, p. 86.

24. P. Smith—Glaucoma, p. 153.
25. Nicati—Soc. Chir., Paris, 1881.
26. Nettleship—Loc. Cit.
27. P. Smith—Glaucoma, p. 158.
28. Rheindorf—Op. Cit.
29. Bailey—R. L. O. H. Rep., IX, p. 199.
30. P. Smith—Glaucoma, p. 153.
31. T. Collins—R. L. O. H. Rep., XIII, 2.
32. Ditto—Lancet, Dec., 1894.
33. Ditto—R. L. O. H. Rep., 1893, p. 502.
34. Snellen—Arch. Oph. N. Y., Jan., 1895.
35. Swanzy—Diseases of the Eye, p. 348.
36. Bourgon—Ann. d'ocul., CIX, 1893, p. 171.
37. Badal—Ann. d'ocul., 1883, p. 231.
38. Stilling—Arch. Oph., XVI, p. 445.
39. Laqueur—Arch. f. Oph., XXIII, p, 149.
40. Manthner—Arch. Oph. N. Y., VII, 1878, p. 242.
41. Noyes—Trans Am. Oph. Soc., 1889, p. 287.
42. Galezowski—Bull. de l'Acad. de Med., Paris, XXXI, p. 240, 1894. Rep. by Chauvel.
43. Abadie—Quoted in Lancet, 1897.
44. DeWecker—Ann. d'ocul., Oct., 1896.
45. Nettleship—R. L. O. H. Rep., XII, 1888, p. 72.
46. Stedman Bull—Trans. Am. Med. Soc., 1889.
47. Gruening—Ditto, 1889, V, p. 287.
48. T. Collins—R. L. O. H. Rep., XIII, 2.
49. Zentmayer and Posey—Op. Cit.

CHAPTER XV.

METHODS OF OPERATING.

IRIDECTOMY; ANTERIOR SCLEROTOMY; NICATI'S SCLEROTOMY; KNIES' MODIFIED IRIDECTOMY; VINCENTIIS'S OPERATION; EXTRACTION OF THE LENS; INTRA-OCULAR MYOTOMY OR HYPOSCLERAL CYCLOTOMY; EXCISION OF NASAL NERVES; ENUCLEATION; EVISCERATION; AND OPTICO-CILIARY NEUROTOMY OR NEURECTOMY.

Iridectomy.

It happens not infrequently that the eye is too hard and too congested to absorb cocain sufficiently to dull the sensibility, especially of the iris, and a general anæsthetic is then required. When an iridectomy is about to be performed for glaucoma, it is well to have the eye not to be operated upon under the influence of eserin, as was suggested by Arlt, in order to diminish the risk of a glaucomatous attack in it; and when it has been decided to employ cocain, which answers perfectly in the less congested forms of the disease, the same eye should have its sensibility diminished by the instillation of a weak solution which will not counteract the desired miosis. The use of eucain, which is not a miotic, though it may cause congestion, is probably attended by little danger of producing glaucoma. Before proceeding to the operation, the surgeon will ask himself what it is which he wishes to accomplish. We have already seen what are the probable methods of cure after iridectomy, and the usual causes of failure. His purpose, then, is the re-establishment or formation of a means of exit for the intra-ocular fluid, and his first aim should be to open the filtration angle by removing the iris up to its utmost periphery. This he can do in such cases as have not lasted sufficiently long for firm adhesion to have been set up between iris and cornea, and where the iris is in a fairly healthy condition. Where the adhesion has become secure we cannot hope for good results, unless by the formation of a permanent filtration tissue, communicating with either the anterior or posterior chamber, and these, as we have seen, when successful, are lined by atrophied iris or ciliary structure, a condition dangerous for the future health of the eye; and yet, as

Treacher Collins has said (1), under these circumstances a sclerotomy producing such a permanent gap would give the best possible results. The removal of a portion of the iris being the primary object the surgeon should select the upper part, because there the coloboma may be hidden by the lid; but if any other segment of the iris appears likely to give a better result, that should be preferred, and this better part may sometimes be recognized by the shape of the pupil which is apt to be drawn up where the iritic adhesion is greatest, as well as by the ectropion of the uveal pigment and the shallowness of the anterior chamber, which will be most noticeable in a like position. (Brailey 2, Treacher Collins 3). The incision need not be very peripheral, and the corneo-scleral junction or a little outside of it will do, but it should be made slowly and of considerable length, perhaps one-fifth of the circumference, to give sufficient room for the next step and to add to the chance of including in the removed portion a non-adherent part of the iris. The choice of a knife will vary with the predilection of the operator, but on the whole, a narrow Graefe is probably the best, great care being taken in any case to avoid the lens, and with a Graefe not to saw upon it through the iris with the back of the knife. Treacher Collins (4) has shown that iridectomy cicatrices are always oblique, and especially so when a keratome is used. In 23 eyes examined by him, only two incisions had touched the ligamentum pectinatum, and in one of these the lens escaped and the ciliary body prolapsed next day. In none was Schlemm's canal reached. Most were corneal and scleral combined. Experimentally he found that an incision 2 m.m. from the cornea, the knife cutting perpendicularly out, passes through the central part of the ligamentum pectinatum, and in absolute glaucoma also cuts the iris at puncture and counter-puncture and passes through it as the incision is completed. Opposite the ligamentum pectinatum the anterior ciliary processes lie close behind the iris, protecting the lens, but if the incision be made here, the processes will prolapse. It must, therefore, not be made so peripheral as the spaces of Fontana.

When the anterior chamber is very shallow and the globe hard, preliminary posterior sclerotomy, as practiced by Priestley Smith and others, may be useful in diminishing the risk of wounding the iris and lens, and of intra-

ocular hemorrhage. When a Graefe's knife has been inadvertently passed through the iris, it is frequently recommended that the incision be completed as usual, but this is doubtful wisdom, as a divided iris cannot be removed so completely as one which is intact, and it would be better to withdraw the knife to be re-entered, either immediately or only after the refilling of the anterior chamber, according to circumstances. The incision having been completed very slowly, so as to lessen the tension as gradually as possible, some surgeons like to allow a solution of cocaine to enter the anterior chamber along the concavity of the curette in order to diminish sensibility in the iris. The iris forceps should then be passed into the wound, the iris carefully seized close to its pupillary edge and as far to one side as possible, pulled out, snipped vertically on the side of the forceps far from the center of the wound, then by passing the forceps slowly, but firmly, quite to the other end of the corneal incision, the iris is torn away at its thinnest part, its origin from the ciliary body, and not, as is sometimes affirmed, cut by the sharp edge of the corneal wound. This operation differs in essential details from the usual iridectomy, in which the iris is seized and snipped off, with the necessary result that a piece is left behind, and Treacher Collins (5) has proposed to accentuate this difference by resuscitating for it the old term "iridectomedialysis." The tearing away of the iris is the only way to remove it up to its very periphery, and is attended by little or no more hemorrhage than occurs with the use of the scissors.

The edges of the coloboma of the iris are, if necessary, now replaced with a spatula, the lens being carefully avoided, and the eye, or better at first both eyes, bound up. Eserin should never be used until the wounds are healed and the eye has quieted down, for before that stage its chief action is to irritate with considerable probability of the production of iritis.

The full effects of the operation on vision are not generally apparent until some time, perhaps two or three weeks later, when the wounded surfaces have healed, but the amount of the resulting astigmatism remains indefinite for a longer time, owing to the continual contraction of the cicatrical tissue. When both eyes are glaucomatous, the question will arise as to the wisdom of operating upon them at the same time. In acute cases, in which it is im-

portant to reduce tension immediately, this should certainly be done, trusting to careful precautions for the avoidance of possible accidents. But when a little delay can make no practical difference, and where other circumstances do not interfere, it is always better not to expose both eyes to possible risk from infection, blows, etc. Schweigger has said (6): "Experience teaches us that in all cases of chronic glaucoma, it is advisable to operate first upon the worse eye, even if it be absolutely blind. Should this be followed by the normal healing process, the second eye may be operated upon without the least apprehension." This may be good advice where the patient is certain to submit to the second operation, but it is probable that, in spite of the most thorough explanation, the first would deter a certain proportion of patients from another operation. Nettleship prefers to begin on the better eye (7). Again, we may well inquire whether it would not sometimes be advisable to operate on a perfectly sound, though *predisposed eye*, whose fellow is already glaucomatous. The answer would seem to depend on circumstances. If the patient is acquainted with the premonitory symptoms, and is within easy reach of a reliable surgeon, the sound eye should be left intact; while operation should be considered when circumstances point to a serious risk of confirmed glaucoma before relief could be obtained. And eserin at least, should always be within the patient's easy reach. Treacher Collins (8) has stated his belief that it is sometimes justifiable to make an iridectomy for preventive purposes, and quotes v. Graefe (9) as asserting that "glaucoma simplex affects almost without exception both eyes successively." Priestley Smith (10) declares that "in the large majority of cases, primary glaucoma is a bilateral affection," and Nettleship (11) that in "something like two-thirds, the disease is sooner or later symmetrical."

Certain *accidents* are liable to happen during or after an operation for glaucoma. Intra-ocular hemorrhage is one of the most frequent, and may have disastrous effects upon the tissues of the eye. The blood is usually absorbed in six or eight weeks, but owing to degenerative tissue changes and too slow circulation in the ocular fluids, it may be delayed even longer. Glaucoma malignum has already been discussed. Where high pressure coexists with a weakened zonule, the lens is in danger of displacement on the escape of the aqueous. A previously clear

lens may be found when close examination of the eye is first possible, to be in a state of more or less advanced cataract. This is said by some authorities to be in every case the result of contact with the edge or back of the knife, with the forceps, or the spatula. It may be that this statement is correct, but experience justifies one in doubting its accuracy, while sudden changes in ocular conditions due to the operation might theoretically be as capable of affecting the lens as say, friction on it through the cornea, which is usually believed to possess the power of ripening cataract. For example, it is to the writer's mind very doubtful whether, especially in advanced age the lens is capable of absorbing its normal amount of nutriment from fluids which, while the scleral incision remains open, pass from the eye nearly as soon as they are formed, and besides may not in such circumstances remain of their usual constitution. We have already seen how Nicati and Greef have proved important changes in the aqueous after a simple puncture of the anterior chamber.

In no inconsiderable number of apparently healthy senile lenses where the pupil is well dilated peripheral striæ may be found, and Zentmayer and Posey have noted lenticular opacities in 95 out of 165 eyes suffering from simple glaucoma, or about 60 per cent. Bull (12) reports that iridectomy materially hastened the growth of opacity in the lenses of 20 out of 35 glaucomatous eyes with previous peripheral striæ. When the capsule is actually cut or ruptured, and the lens, absorbing fluid begins to swell, a condition arises in itself liable to cause glaucoma, and very different from that in which there are merely a few striæ to mark the approaching opacity. In the former case the eye is said by Fuchs (13) to be almost certainly lost, and at the best is in considerable danger, for the circumstances are anything but propitious for the extraction of the offending lens.

Anterior Sclerotomy.

In Quaglino's original sclerotomy the scleral incision was made continuous from end to end, but Wecker changed this, in order to diminish the great tendency to iritic prolapse, by leaving a bridge of tissue uncut and his operation, or some slight modification of it, is the one which is still generally in use. But before deciding to

operate it should always be ascertained that the iris will react well to a miotic, under whose influence it should very distinctly be at the time of the operation. Otherwise the operation should be avoided. The necessary local or general anæsthetic having been employed, and the second eye being also in a state of miosis, and if cocain be employed also partially insensible, a narrow Graefe's knife is made to puncture, as if for cataract extraction, 1 m.m., however, behind the corneo-scleral junction; but as soon as it enters the aqueous it is, on account of the shallow anterior chamber, directed upward, so as to follow closely the angle of the anterior chamber, insinuating itself slowly around to a point on the other side of the globe opposite the puncture, when it is passed through the sclera, and with a sawing motion the incision is slowly made at the original distance from the cornea till a piece of sclera only some 3 m.m. wide is left uncut, the knife being then withdrawn carefully and with slight pressure forward, in order to completely evacuate the aqueous humor. A subsequent deviation from the circular in the form of the pupil *shows a tendency to a prolapse* of the iris, and if it occur and the iris cannot be gently replaced with a spatula, or even iris forceps, the sclerotomy will have to be turned into an iridectomy. Eserin should be again used, both eyes bandaged, and an examination for prolapse made two or three hours later. The patient should be on his back till the wound is fairly well united. Sometimes a second sclerotomy, or a sclerotomy following an iridectomy, is necessary, and it is then usually made directly opposite the former point; while in some cases, such as at times in serous iritis, it may be necessary to repeat the operation several times, in which case the best method is to use a keratome, making a single incision on each occasion.

Nicati's Sclerotomy.

Nicati (14) has published particulars of an operation which he calls *sclero-iritomy*, whose object is to "place the posterior chamber in direct communication with the meshes of the subconjunctival areolar tissue, and at the same time modify the secretion of the aqueous humor by dividing the nerves of the iris;" and also, he adds later, to "diminish the reflex contractions of the tensor muscle of the cho-

roid," which he considers bear an important part in the production and regulation of the ocular tension. "The puncture, which is made just outside the cornea, penetrates the iris to enter the posterior chamber where the lens is avoided by means of a carefully calculated procedure. The knife, a lancette à arret, is passed under a fold of the conjunctiva in such a manner as to insure a valvular opening at a tangent to the cornea and perpendicular to the plane of the iris." He sums up the result of 57 of these operations by saying (15) that *(a)* "they are remarkably favorable in the treatment of staphyloma following necrosis of the cornea, and especially as a preventive measure against this affection; and *(b)* that the result is mediocre in ordinary glaucoma, on account of the tendency to hyphæma, and because, the incision being perpendicular to the coverings of the eye, the edges of the wound are not separated by alternations of pressure, as they are in an oblique wound formed in an ordinary sclerotomy. The incision tends, consequently, to heal quickly without leaving any curative fistula." In 1894 Nicati (16) asserted that this remained the preventive and curative operation for cicatrical staphyloma of the cornea and cicatrical glaucoma. This operation he *modified* for use in ordinary spontaneous glaucoma, performing it with a sharp, narrow knife. "First stage (intruduction): The blade, edge downward, is introduced through the sclerocornea into the inferior angle of the anterior chamber, placed parallel to the iris in a horizontal direction, and finally pushed along in such a manner as to pierce the sclerotic a second time, beyond which counter-opening the point should be made to project 1 c.m. This corresponds to the first stage of Wecker's sclerotomy. Second stage (rotation)—Giving the blade a quarter turn, it is placed perpendicularly to the iris, with which membrane the edge now lies in close contact. By this maneuver an incision perpendicular to the first is made in the sclerotic and the aqueous humor escapes. Third stage (extraction)—The blade is rapidly withdrawn in the same plane, as in the second position. In doing this the iris is divided along its line of attachment to an equal extent to the contact with the edge of the knife. It goes without saying, that care should be taken to remove all that is possible of the blood poured into the anterior chamber as a result of the section of the iris. As far as that is concerned, however, the de-

pending portion of the wound favors the spontaneous evacuation of the blood. This operation fulfills the indication which requires that the scleral wound should gape easily, since not only is the incision not perpendicular to the sclerotic, but by the second step of the operation it is transformed into a genuine V shaped flap, a form of wound that insures the flow of the aqueous humor better than any other. The operation fulfills also, the indication of dividing freely the nerves of the iris, constituting a thorough iridian neurotomy, which is capable of diminishing the secretory impulses reflected from the membrane. It obeys the call for a good glaucoma operation, such as we have established it to be in a physiological aud pathological study of the glands of the aqueous humor." (See Nicati's theory). He sums up this procedure by saying, that it "becomes the operation of choice for spontaneous glaucoma." Nicati has not yet published his results by this operation, but Monk (17) has given a few particulars concerning them, insufficient, however, to enable the reader to come to an independent conclusion regarding the worth of this operation as compared with iridectomy. The number of operations was then 22, and included acute, chronic, primary, and secondary cases. "Experience teaches that care should be taken to divide the iris thoroughly, and that in order to carry out this object, the knife should not be too narrow. It is even permissible to ensure the section of the iris by a few backward and forward movements of the knife in its last position, just before withdrawal." The second scleriritomy, according to Nicati, has limited the value of iridectomy to cases of glaucoma with occlusion of the pupil.

Knies' Modified Iridectomy.

Knies in 1893 (18) reported having in two cases cut the base of the iris in making the incision. One case was annular posterior synechia, and the other primary acute glaucoma. Eserin should be used before the operation, and a Graefe knife should be employed. One should cut slowly, and may leave a bridge of sclera or conjunctiva. It is not very easy on account of possibly wounding the lens or zonule; "but the anterior chamber is shallow and at first—at least—the lens is not pushed forward to a corresponding extent." Priestley Smith says "advance of

the iris depends on the position of the lens," this operation being therefore dangerous. Knies calls it *iridо-sclerotomy*.

As has been said, Treacher Collins, in 1891, had already advocated the incision of the base of the iris with the object of opening up a new exit from the anterior chamber, in certain cases in which iris and cornea are adherent (19). The following, written by him at the same time, may advantageously be read in this connection: "If the root of the iris has become adherent to the cornea over the region of the ligamentum pectinatum, as so often is the case in chronic glaucoma, then, if an incision passes across the ligamentum pectinatum, the iris must necessarily be wounded; and if the root, as is not unusual, is atrophied as well as adherent, it would be almost impossible to wound it without penetrating it. That, in iridectomy, incisions situated internal to the commencement of the ligamentum pectinatum, and made with either a Graefe's knife or a keratome, divide the root of the iris is shown by (certain) cases, in both of which there was a wound in the lens capsule, opposite the incision in the sclero-corneal tissue. If, in making a sclerotomy incision, the knife passes into the posterior chamber opposite the ligamentum pectinatum, there is less likelihood of its wounding the lens than if it enter less peripherally, for the anterior of the ciliary processes are immediately behind the iris in this region and serve to protect the lens. Moreover, the lens does not extend as far outward as the commencement of the ligamentum pectinatum."

Vincentiis' Operation.

The following operation had been employed by Vincentiis (20) in various forms of glaucoma more than 70 times, and by his assistants 10 times. He says: "I propose to execute simple separation of iris from cornea at the angle of the anterior chamber by the aid of an instrument whose use is limited to the unplaiting of tissue, or to disengagement or even tearing at the iritic angle. What I can assert is that I have performed the said operation 4 times in absolute glaucoma, and 4 times I have obtained a result truly marvellous, because, without any irritative symptom having ever shown itself, except a slight injection of the episcleral tissues, as one observes it after the incision of the tissue of the iritic angle, the ocular tension has fallen at

the end of 24 hours to the normal state, and there remained during more than the week during which the patient continued under our observation. The instrument with which I have practised this disengagement of the angle of the anterior chamber is analogous to that with which I make the incision of the iritic angle,* as well regarding the size of the blade, which is proportionate to its cutting end, as the slightly oval form of that end." It somewhat resembles a small crochet needle, blunt at its convexity and sharp at the end of its concavity. It is so made that "when the instrument has penetrated through the sclera 1.5 m.m. from the corneo-scleral border and has entered the anterior chamber from whatever direction one reaches with it the iritic angle, with the point in front, or behind which is preferable, it will always be the convex part which will find itself in contact with the iritic angle." The pressure on the angle draws away the pupil to that side and indicates the amount of force employed. Vincentiis believes that iridectomy will remain the operation in acute glaucoma, for by it one can obtain the most extensive modifications in the tissue of the iritic angle, which, on account of the corneal opacity and of the narrowing of the anterior chamber, can be directly produced by neither the incision of the tissue of the iritic angle nor by sclerotomy.

To diminish high tension Vincentiis would place in the first rank iredectomy, then incision of the iritic angle, and then sclerotomy. He would put his operation in the place of sclerotomy wherever the latter is now used, for, besides its better results, it shows a greater regard for the ocular tissues, the permanent integrity of the anterior chamber, and the absence of all immediate alteration in the equilibrium of the ocular tension, with all the useful results which flow from that.

Extraction of the Lens.

MacKenzie (21) found that extraction of the lens was sometimes of use, but he did not recommend it for general adoption, because he had seen such violent inflammation follow.

Rheindorf (22) wrote a paper advising this operation in malignant glaucoma, and added an incision into the "vitreous depression;" he also advised its adoption in cases

*A diagram of this is given by Vincentiis' first assistant, Ugo Tailor, on page 4 of his paper "Sulla Incisione dell 'Angulo Irideo," Napoli. Tipografia Angelo Trani, Strada Medina, 25, 1891.

where iridectomy and sclerotomy had had no good results, as well as in absolute cases in place of enucleation. In reply to the criticism that one cannot remove the lens in glaucoma with benefit to vision, he replied that the prospects for the eye are improved by this procedure, (A) when after iridectomy the anterior chamber is not reformed, *(a)* in acute cases relatively soon after, and *(b)* in chronic inflammatory and simple cases when the vision falls in the course of a few days; (B) when after iridectomy the anterior chamber reforms, but the vision further decreases; *(C)* in absolute glaucoma instead of enucleation. In 40 operations he had never seen intra-ocular hæmorrhage. On this subject Nettleship (23), who considered malignant glaucoma to be sometimes due to a weak suspensory ligament allowing the lens to become dislocated forward, thought that if it were not that in these cases the vessels are also generally so unsound as to be liable to rupture, the best treatment would be extraction of the lens in its capsule, and even with that risk it might sometimes be a good operation. From the importance which Priestley Smith has attached to the lens in the pathology of glaucoma one would expect him to advocate its removal in the capsule in certain cases, but he says (24), "the practical surgeon would shrink from attempting an extraction under such circumstances. The lens being healthy and sometimes disproportionately large, would demand a larger incision than is required for the cataractous lenses which we commonly extract, and it would separate from its attachments with greater difficulty."

When the lens has to be removed on account of cataract, there being reason to believe that there is a sufficiently healthy condition of the fundus, it is probably best to reduce tension first by an iridectomy and extract some weeks later.

Intra-Ocular Myotomy or Hypescleral Cyclotomy.

This operation was performed on the supposition that cramp of the ciliary muscle caused a stasis in the intra-ocular blood circulation. It was employed by Hancock in 1860, Heiberg in 1862, Pritchard in 1871, and Ortowski in 1872 (25). Hancock inserted Beer's knife at the outer lower scleral border and passed it down and back till the sclera showed a 3 m. m. section; or, after inserting it at the limbus and through the angle of the anterior chamber

the muscle was cut "with the lens on one side and the sclera on the other side of the knife" (26). Autopsies showed that many of these incisions had divided a part only of the muscle.

Ciliary Punctures.

The object being not the division of the muscle, but rather to open the aqueous and vitreous chamber at the same time, have occasionally been employed with more or less success. Their action on tension, unless by the possible displacemont of the iris in recent cases, or by the dangerous inclusion of iris and ciliary process in the wound, must be less lasting than that of iridectomy, and for these reasons it is not an operation to be recommended.

Excision of the Nasal Nerves.

On the ground of Donders' hypothesis, that glaucoma is due to a neurosis affecting secretion, Badal and Lagrange (27) stretched and excised the infra-trochlear branch of the fifth nerve, and asserted that the results were reduction of pain and tension, with improvement, even in cases in which iridectomy and sclerotomy had been of no service. The practice, however, seems to have fallen, as might be expected, into complete disuetude.

Enucleation, Evisceration, and Optico-Ciliary Neurotomy or Neurectomy.

Instead of removing the globe in cases where the eye is both blind and painful, the surgeon sometimes prefers to eviscerate, by removing the cornea and then cleaning out the entire contents of the sclera, which is left free of any signs of uvea. An artificial glass or silver vitreous (Mule) is sometimes placed within the sclera before it is sewn up. Or, the globe having been removed, the ball has sometimes been sewn up in Tenon's capsule. All of these operations are superior to enucleation, so far as a movable foundation for an artificial eye is concerned, but the former cause a more prolonged and painful convalescence. From cosmetic considerations, optico-ciliary neurotomy or neurectomy, sometimes replaces enucleation. The conjunctiva having been divided over the internal or external rectus, the muscle is seized and divided a few m.m. from the globe, after having been secured on the distal side of the point of section by needle and thread. The optic nerve is then divided as far from the globe as possible,

the eyeball is rotated so that the remains of the nerve and all the tissue on its posterior surface may be cut down close to the sclera. The eye is then replaced and the muscles sutured. This operation does not always prevent the return of pain in glaucomatous eyes. In two eyes, each removed for pain, two years after optico-ciliary neurotomy, Treacher Collins (28) found extreme atrophy of the uveal tract and retina, but less marked in front of, than behind, the equator. The lenses were clear.

An eminent London ophthalmic surgeon has informed me that he has in several cases of blind glaucomatous eyes *scraped back all the iris*, and even the ciliary body, from proximity to the cornea, and left them, with the result that the tension has fallen to normal and the pain has disappeared.

It is erroneously held by some surgeons that a glaucomatous eye cannot give rise to sympathetic ophthalmia, because the channels at the back of the eye are closed. Even if that were true, conditions may be altered when the tension has become normal after operation, and sympathetic ophthalmia is not unknown in glaucomatous eyes, even after optico-ciliary neurectomy and evisceration (29).

REFERENCES CONTAINED IN CHAPTER XV.

1. Treacher Collins—R. L. O. H. Rep., XIII, 2.
2. Brailey—R. L. O. H. Rep., IX, p. 224.
3. Treacher Collins—Loc. Cit. (1).
4. Ditto.
5. Treacher Collins—Lancet, Dec. 1894.
6. Schweigger—Arch. Oph., N. Y., XX, 1891, p. 475.
7. Nettleship—Brit. Med. Ass., 1895.
8. Treacher Collins—R. L. O. H. Rep., XIII, 2, Dec., 1891, and Lancet, Dec. 1895, p. 1467.
9. v. Graefe—R. L. O. H. Rep., VII, p. 104.
10. Priestley Smith—Glaucoma. 1879, p. 5.
11. Nettleship—R. L. O. H. Rep., XII, p. 79.
12. Bull—Trans. Am. Med. Ass., 1889.
13. Fuchs—Augenheilkunde, p. 402.
14. Nicati—Soc. de Biologie, July 4, 1891, and Ass,. Fran. pour l'avanc. de sciences Con. de Marseille,, Sep.. 1891, and Bull, et Mem. de la Soc. Fran. d'Oph., May 4, '92.
15. Nicati—Ann. d'ocul., Sep., 1893.
16. Nicati—Rev. gen. d'oph., Jan., 1894, and Monk in Oph. Rev., Lond., April, 1894, p. 123.

17. Monk—Loc. Cit. (16).
18. Knies—Heidelberg Oph. Soc., Aug., 1893, Rep., p. 118, and Priestley Smith, Oph. Rev., Lond., XIII, p. 24.
19. Treacher Collins—R. L. O. H. Rep., XIII, 2.
20. Vincentiis—Rev. gen. d'Oph., Nov. 30, 1894.
21. MacKenzie—Dis. of the Eye.
22. Rheindorf—Klin. Monatsbl. f. Augenh., XXIX, p. 33, 1891.
23. Nettleship—R. L. O. H. Rep., XII, 1888, p. 72.
24. Priestley Smith—Glaucoma, p. 162.
25. Mauthner—Arch. Oph., N. Y., VII, 1878, p. 178.
26. Koenigstein—Wien. Med. Blaet., XV, 1892, p. 773.
27. Badal and Lagrange quo. by Koenigstein, Loc. Cit. (26).
28. Treacher Collins—R. L. O. H. Rep., XIII, 2, 1891.
29. Fuchs—Augenheilkunde, p. 804.

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